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Cardiovascular risk factors among young adults.

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**CARDIOVASCULAR RISK FACTORS
AMONG YOUNG ADULTS**

by

Linda May Stanczak

**A Thesis
submitted to the
Faculty of Graduate Studies and Research
through the Department of
Kinesiology in Partial Fulfillment
of the requirements for the Degree
of Master of Human Kinetics at
the University of Windsor**

Windsor, Ontario, Canada, 1988

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ABSTRACT

CARDIOVASCULAR RISK FACTORS AMONG YOUNG ADULTS

by

Linda May Stanczak

Cardiovascular risk factors were measured among 56 young adult subjects (age 21-32 years), half of whom had a parental history of CHD and half who did not. The dependent variables (risk factors) included total serum cholesterol, LDL cholesterol, HDL cholesterol, triglycerides, smoking, blood pressure, body composition, diet, and physical activity. Multivariate analyses failed to show differences in several dependent variable combinations. One way analyses of variance revealed a significant difference ($p < .1$) only in triglyceride levels among young adults with or without parental history of CHD. Several significant correlation coefficients ($p < .1$) were computed among the dependent variables and their component parts although few revealed an explained variance of greater than 25%.

This study is in partial agreement with numerous other investigations which document the tendency for first-degree relatives of CHD patients to have more adverse risk factors than the general population. This trend has also been shown among children and adolescents although the differentiating factors are inconsistent. The actual effect of elevated triglyceride levels in the cardiovascular risk factor profile is unclear from an epidemiological viewpoint although elevated triglycerides are commonly treated in clinical practice.

DEDICATION

to my husband, Ray,
for his encouragement and especially for his patience

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TABLE OF CONTENTS

Abstract	iv
DEDICATION	v
ACKNOWLEDGEMENTS	vi
 Chapter I: INTRODUCTION	 1
 Chapter II: REVIEW OF LITERATURE	 3
Plasma Lipids and Lipoproteins	3
Diet	6
Smoking	11
Blood Pressure	12
Physical Activity	15
Body Composition	19
Family History	22
Occupation/Education	24
Summary	25
 Chapter III: METHODOLOGY	 26
Statement of the Problem	26
Hypotheses	26
Definitions	28
Assumptions	29
Limitations	29
Delimitation	30
Selection of Subjects	30
Experimental subjects	30
Control subjects	31
Both subject groups	31
Independent and Dependent Variables	31
Independent Variable	31
Dependent Variables	31
Testing Protocol	36
Statistical Analyses	37

Chapter IV: RESULTS	39
Chapter V: DISCUSSION	47
Chapter VI: CONCLUSIONS AND RECOMMENDATIONS	56
Appendix A: CONSENT TO TESTING PROCEDURE	58
Appendix B: YOUNG ADULT QUESTIONNAIRE	59
Appendix C: OUTLINE OF INSTRUCTIONAL SLIDE PRESENTATION FOR DIET REPORT	62
Appendix D: 3 DAY DIET RECORD INSTRUCTIONS	63
Appendix E: DIET REPORT FORM	66
Appendix F: PARENT INFORMATION	67
Appendix G: PHYSICIAN LETTER	71
Appendix H: PHYSICIAN VERIFICATION OF MEDICAL HISTORY	72
Appendix I: YOUNG ADULT SCREENING QUESTIONNAIRE	73
Appendix J: RAW DATA	77
Appendix K: SIGNIFICANT CORRELATION COEFFICIENTS ($p < .1$)	82
BIBLIOGRAPHY	84
VITA AUCTORIS	103

LIST OF TABLES

1.	Lipoprotein Changes With Diet	9
2.	Blood Pressure Score	32
3.	Body Composition Score	33
4.	Physical Activity Score	35
5.	Conversion of raw diet data into diet scores	36
6.	Physical characteristics of young adult subjects.	40
7.	Parent Characteristics	41
8.	MANOVA results based on parental history	41
9.	ANOVA results based on family history	42
10.	ANOVA results based on gender	44
11.	Physical activity of CHD parents	50

LIST OF FIGURES

1.	Smoking interaction	45
2.	Systolic blood pressure interaction	46

Chapter I

INTRODUCTION

Cardiovascular disease is the leading cause of death in most western countries. Coronary heart disease (CHD) resulting from the accumulation of atherosclerotic deposits in the coronary arteries kills almost 50,000 people per year in Canada (Nicholls, Nair, MacWilliam, Moen, & Meo, 1986). Financially, the cost of this disease is large. This includes the direct expenses of health care, hospitalization, and pharmaceutical products, as well as the loss of work productivity. Obviously, a reduction in the amount and severity of CHD would provide many widespread benefits.

Pathologic changes which lead to atherosclerosis begin in infancy and progress during childhood making heart disease not only a disorder of the middle-aged and elderly but also a pediatric problem (Kannel & Dawber, 1972). Enos, Holmes, and Beyer (1953) and McNamara Molot, Strempel, and Cutting (1971) were able to show significant degrees of atherosclerosis among young war casualties who had no apparent evidence of heart disease prior to autopsy.

Epidemiological studies have established the risk-factor concept as a means of predicting morbidity related to CHD. Cigarette smoking, serum lipid values, and blood pressure have been clearly identified as critical factors in predicting an individual's risk for developing CHD. Of great importance is the notion that these three factors can be modified to some extent. Other factors which have been identified include physical activity habits, dietary practices, and body composition, all of which can be altered. On the other hand, age, gender, and family history are fixed.

Much of the current knowledge regarding the influence of modifiable, lifestyle-related factors in the development of coronary risk factors comes from longitudinal studies of middle-aged and older-aged adults. Lifestyle patterns are generally well established among such populations and favourable changes in behaviour are infrequent or are a consequence of perceived ill health or treatment for existing disease (Hubert, Eaker, Garrison, & Castelli, 1987). It is also possible that the atherosclerotic process may be too far advanced in the older age groups to demonstrate clear benefits from risk factor intervention. Although data are accumulating on risk factor levels among children and adolescents, few studies have examined CHD risk factors in young adult populations who may be more likely candidates for appropriate intervention (Donahue, Orchard, Kuller, & Drash, 1985). A greater understanding of the determinants of CHD risk factors in young adulthood would clearly enhance preventative efforts aimed at this age group.

Chapter II

REVIEW OF LITERATURE

2.1 *Plasma Lipids and Lipoproteins*

The process of atherosclerosis and its clinical effects of CHD can be viewed as a disorder in lipid metabolism (McMurray, 1977). The lesions consist of a deposition of lipids largely composed of cholesterol. As a result, much of the research in the development of CHD has focused on the levels of serum cholesterol, its lipoprotein subfractions, and triglycerides (TG).

A graded association of plasma cholesterol level with coronary risk has been a consistent finding in epidemiological surveys of many populations (Carlson & Bottiger, 1972; Dawber, 1980; Keys, 1980; Pooling Project Research Group, 1978; Rosenman, Brand, Jenkins, Friedman, Strauss, & Wurm, 1975; Stamler, 1979). The Pooling Project utilized data collected from several similar epidemiological studies in the United States. No significant differences in CHD rates among men aged 40-64 years were found between the lower two quintiles of total-plasma cholesterol levels (<5.66 mmol/l). The relative risk of those in the upper two quintiles (>6.18 mmol/l) was 2.0 to 2.4 times that of men in the lower two quintiles. These differences in risk with increasing levels of serum total cholesterol were more pronounced in the younger (40-44 years and 45-49 years) age groups. Women showed a similar association although the gradient was not as great (Pooling Project Research Group, 1978).

Several investigators have attempted to determine the ideal level of total plasma cholesterol below which a change in CHD risk is not evident. Its relationship with the risk of CHD appears to be curvilinear with a threshold level in the range of 5.17-5.69 mmol/l (Pooling Project Research Group, 1978). As a guideline, a population average of 5.17 mmol/l is likely to be associated with no more than a moderate frequency of CHD (World Health Organization, 1985). Thus, desirable levels would be below this range. This conclusion is consistent with another proposal that the ideal level of total cholesterol for adults would be in the range of 3.36-4.91 mmol/l (Stamler, 1979).

Discrimination of those at risk is substantially improved when total plasma cholesterol is fractionated into its lipoprotein components. Low density lipoprotein cholesterol (LDL) appears to be the most atherogenic. Studies have shown it to be an independent factor in the development of CHD (Gordon, Castelli, Hjortland, Kannel, & Dawber, 1977a, 1977b; Illingworth & Connor, 1985; Wilson, Garrison, Castelli, Feinleib, McNamara, & Kannel, 1980). A direct relationship between LDL concentrations and the extent of coronary atherosclerosis has been shown in angiographic studies (Jenkins, Harper, & Nestel, 1978; Naito et al., 1980). Leon (1987) suggested that a level of LDL greater than 3.36 mmol/l is an important risk factor.

It has been found in cross-sectional studies that persons with documented CHD generally have lower levels of plasma high density lipoprotein cholesterol (HDL) than do persons without the disease (Assmann, Schultf, Oberwittler, & Hauss, 1986; Barr, Russ, & Eder, 1951; Castelli et al., 1977; Miller, 1986; Miller & Miller, 1975). Low levels of HDL have also been implicated in men who were considered to be at relatively low risk according to several well documented factors but who subsequently developed CHD (Heller, Miller, Wheeler, & Kind, 1983). The ratio of total cholesterol to HDL cholesterol levels appeared to be a better predictor of CHD events than either lipid component alone (Kannel, 1983, 1986).

HDL particles are quite variable in size, density, lipid, and apoprotein composition and thus have been divided into several subclasses (Alaupovic, 1984). It appears that the low HDL cholesterol levels in those with clinically significant CHD reflect reductions of both HDL₂ and HDL₃ and that the reduction of HDL₂ tends to be proportionately greater than HDL₃ (Ballantyne, Clark, Simpson, & Ballantyne, 1982; Hamsten, Walldius, Dahlen, Johansson, & DeFaire, 1986; Wallentin & Sundin, 1985). The question of whether measurement of HDL₂ is superior to that of HDL cholesterol in the assessment of heart disease risk has not yet been answered (Miller, 1987).

In an early study, Albrink and Man (1959) proposed that triglycerides (TG) may be causal in the development of CHD when high levels of TG were found in men with a history of myocardial infarction (MI). The basic epidemiological association of elevated TG and increased incidence of CHD has been confirmed in many studies (Carlson & Bottiger, 1972; Castelli et al., 1977; Hamsten et al., 1986; Pelkonen, Nikkila, Koskinen, Penttinen, and Sarna, 1977; Rhoads, Blackwelder, Stemmermann, Hayashi, & Kagan, 1978; Robertson et al., 1977; Rosenmann, Brand, Sholtz, & Freidman, 1976) but its biologic basis is uncertain. The consistency of the unadjusted association in so many studies makes it unlikely that the association is artifactual but it does not permit the conclusion that elevated TG are a cause of CHD (Hulley, Rosenman, Bawol, & Brand, 1980).

Some studies have demonstrated an independent association (Carlson & Bottiger, 1972; Hamsten et al., 1986; Pelkonen et al., 1977). Other evidence has not shown TG to be an independent risk factor when multivariate analysis was used (Castelli et al., 1977; Rhoads et al., 1978; Robertson et al., 1977; Rosenmann et al., 1976). Few studies investigating the TG/CHD relationship have included women. It appears, however, that elevated TG levels are predictive of future CHD mortality in white women over the age of 50 years (Heyden, Heiss, Hames, & Bartel, 1980; Kannel, Castelli, Gordon, & McNamara, 1971; Lapidus, Bengtsson, Lindquist, Sigurdsson, & Rybo, 1985). Even if TG are not an independent risk factor, it may still be an indirect cause of CHD by operating through one or more other risk variables (Hulley et al., 1980).

Very low density lipoprotein cholesterol (VLDL) carries the largest amount of TG and plays an important role in the production of other blood lipoproteins and their transport. Its relationship to CHD, however, is uncertain (Blackburn, 1980).

The blood lipids that have been identified as being important in the development of CHD are all modifiable to some degree. Changes in these levels can be related to the risk of CHD. For every 1% reduction in total cholesterol, CHD incidence can be reduced by 2% (Lipid Research Clinic Program, 1984). Similarly, as little as a 0.26 mmol/l increase in HDL cholesterol level has been associated in observational studies with as much as a 50% reduction in CHD risk (Leon, 1987).

2.2 Diet

Dietary components appear to influence the risk of developing CHD. The Seven Countries Study was a cross-population investigation of men from European countries, Japan, and the United States. The data showed a positive correlation between the proportion of calories consumed as saturated fat and CHD deaths ($r=.84$). A similar relationship was noted between the consumption of saturated fats and serum cholesterol levels (Keys, 1980; Keys et al., 1986). Similarly, the composition of the diet correlated significantly with both serum cholesterol and CHD mortality among Japanese living in Japan, Hawaii, and San Francisco (Marmot, Syme, Kagan, Kato, Cohen, & Belsky, 1975; Robertson et al., 1977). These results concur with many other studies (Armstrong, Mann, Adelstein, & Eskin, 1975; Kozarevic, Pirc, Racic, Dawber, Gordon, & Zukel, 1976; Shekelle et al., 1981; Walden, Schaefer, Lemon, Aunshine, & Wynder, 1964; Wen & Gershoff, 1973). Conversely, some studies have failed to observe significant correlations among dietary fat, serum cholesterol concentration and CHD rates (Dawber, 1980; Nichols, Ravenscroft,

Lamphiear, & Ostrander, 1976). Subjects in these two studies did not fast prior to blood sampling. These negative findings do not necessarily mean that correlations do not exist but are reflective, perhaps, of the genetic heterogeneity and dietary homogeneity in the U.S. population (Grundy et al., 1982).

Dietary intervention trials under controlled, institutionalized conditions have been relatively consistent in the observation that a diet low in saturated fat and cholesterol, and high in polyunsaturated fat can result in a decrease in the serum cholesterol level and a decline in CHD mortality (Dayton, Pearce, Hashimoto, Dixon, & Tomiyasu, 1969; Leren, 1966; Miettinen, Trupezinen, Karvonen, Elosuo, & Paavilainen, 1972; Rinzler, 1968). Although all of these studies have been criticized for flaws in experimental design (i.e., small sample size, lack of randomized control groups), they were uniform in reporting the trend toward lower CHD risk.

Cross-sectional and experimental investigations have generally been in agreement on the effect of diet on lipoprotein levels. Diets with a high content of carbohydrate (65-80% of total energy) and a low fat content (20-25% of total energy) produced a substantial reduction in HDL cholesterol among sedentary subjects (Enholm et al., 1984; Gonen, Patsch, Kuisk, & Schonfeld, 1981; Schaefer, Levy, Ernst, VanSant, & Brewer, 1981). This reduction was mainly the result of a decrease in the HDL₂ subfraction and appeared to be independent of the ratio of polyunsaturated to saturated fatty acids (P/S ratio) (Enholm et al., 1984; Kuusi et al., 1985). LDL cholesterol changes paralleled HDL cholesterol changes, thus the HDL/LDL ratio remained relatively unchanged (Pietinen & Huttunen, 1987).

In diets containing 30-45% of calories as fat, the type of fat appeared to be more important. With few exceptions, HDL cholesterol was decreased in diets with a P/S ratio exceeding 1.5 (Ernst, Fisher, Bowen, Schaefer, & Levy, 1980; Mattson & Grundy, 1985; Schaefer et al., 1981; Turner, Le, & Brown, 1981). Conversely, changes in the P/S ratios between 0.2 and 1.5 have

generally produced no change in HDL (Blaton et al., 1984; Schwandt, Janetschek, & Weisweiler, 1982; Weisweiler, Drosner, Janetschek, & Schwandt, 1983). LDL was consistently reduced in these investigations. The P/S ratio thus played a role in influencing the HDL/LDL ratio in diets containing greater than 30% of the total energy consumed from fat.

Grundy (1986) has recently shown that substituting monounsaturated fatty acids for saturated fatty acids in a diet containing 40% of energy as fat lowered LDL cholesterol as effectively as a low fat diet but without changes in HDL or its subfractions. Unfortunately, the amount of monounsaturated fat in diets is not reflected in the P/S ratio and has not frequently been reported.

There are few long-term studies relating diet to lipoprotein levels. Brown and colleagues (1984) investigated the effects of two "lipid lowering" diets on patients with peripheral vascular disease. The American Heart Association diet of low cholesterol and modified fat (25-30% of calories as fat, P/S=1.7) and Pritikin's maintenance diet of high fiber, low cholesterol and very low fat (5-10% of calories as fat) were used in this 1 year intervention trial (see Table 1). In both diets, favourable lipid changes were observed (decreases in total cholesterol and LDL, and increases in HDL) but changes generally failed to reach a level of significance. Total cholesterol on the Pritikin diet, however, decreased 10% from initial levels and was significant ($p < .01$). After 4 years on a diet consisting of 28% fat with a P/S ratio of 1.0, Norwegian subjects had 20% higher HDL cholesterol levels than control subjects (Hjermann, Enger, Helgeland, Holme, Leren, & Trygg, 1979).

A summary of the lipoprotein changes occurring with modifications in dietary fat can be found in Table 1.

A large proportion of cholesterol in the diet is provided by dairy products and other foods of animal origin. As a result, the effects of dietary cholesterol in epidemiologic studies have been difficult to separate from the effects of dietary fat (Katan, 1984). Clinical trials have generally

Table 1: Lipoprotein Changes With Diet

NS = non-significant
- = not reported

AUTHORS	% calories consumed as fat	P/S ratio	Lipoprotein Changes		
			RDL	LDL	LDL/RDL
Short term studies (< 4 months)					
Gonen et al. (1981)	20-25%	independant	↓	↓	no change
Schaeffer et al. (1981)					
Enholm et al. (1984)					
Schaeffer et al. (1981)	30-45%	≥ 1.5	↓	↓	no change
Ernst et al. (1980)					
Turner et al. (1981)					
Mattson & Grundy (1985)					
Weisweiler et al. (1983)	30-45%	0.2 - 1.5	no change	↓	↓
Schwandt et al. (1982)					
Blaton et al. (1984)					
Longer term studies					
Brown et al. (1984) (1 year)	5-10%	-	↑ (NS)	↓ (NS)	-
	25-30%	1.7	↑ (NS)	↓ (NS)	-
Hjermann et al. (1979) (4 years)	28%	1.0	↑	-	-

lasted no more than a few weeks and almost all have used egg yolks (which contain high amounts of fat) as the dietary source of cholesterol. Usually, egg yolk cholesterol causes an increase in serum cholesterol (Glueck & Connor, 1978; Liebman, 1982) although there are exceptions (Ginsberg, Le, Mays, Gibson, & Brown, 1981). When separate lipoprotein fractions were measured, increases were usually found in both LDL and HDL cholesterol (Nestel, Tada, Billington, Huff, & Fidge, 1982; Raymond, Connor, Lin, Warner, Fry, & Connor, 1977; Schonfeld, Patsch, Rudel, Nelson, Epstein, & Olson, 1982).

A strong positive relationship between alcohol consumption and HDL cholesterol levels has been established in epidemiological surveys (Castelli et al., 1977; Ernst, Fischer, & Smith, 1980; Hulley & Gordon, 1981). It has been estimated that alcohol consumption accounts for at least 4-6% and possible up to 10% of the total variation of HDL cholesterol in Western, industrialized populations (Gordon, Ernst, Fisher, & Rifkind, 1981). Social drinkers have mean HDL cholesterol levels that may be higher than those of abstainers by as much as 30% (Pietinen & Huttunen, 1987). Chronic alcoholics have HDL levels that are even higher.

The fluctuations in HDL cholesterol that have been found with varying levels of alcohol consumption were mainly reflected by changes in HDL₃ rather than in the more atherogenic HDL₂ (Pietinen & Huttunen, 1987). As a result, increasing alcohol consumption has not been a public health measure used in the reduction of CHD (Gordon et al., 1981).

The American Heart Association has made dietary recommendations for the purposes of modifying cardiovascular risk factors, especially high plasma cholesterol (Grundy et al., 1982). Saturated fat intake should be limited to less than 10% of total calories from the current American diet of 15-17%. Clinical studies have shown this reduction in dietary intake to decrease plasma cholesterol by about 0.52 mmol/l (Hegsted, McGandy, Myers, & Stare, 1965; Keys, Anderson, & Grande, 1965). Monounsaturated or polyunsaturated fats are favourable substitutes but the long term effects of polyunsaturates are not known. While some replacement of saturated fats by polyunsaturates seems safe, it may be prudent not to exceed 10% of total calories (Grundy et al., 1982). Monounsaturates do not reduce total plasma cholesterol as effectively as polyunsaturates but they are not likely to be toxic as they are synthesized by the human body in considerable quantities (Grundy et al., 1982). Dietary cholesterol should be limited to a maximum of 300 mg/day (Grundy et al., 1982). Total caloric intake should be adjusted to achieve and maintain desirable weight. These recommendations are consistent with those made by the Intersociety Commis-

sion for Heart Disease Resources (1984), the Concensus Development Conference (1985) and the World Health Organization (1985).

2.3 Smoking

Several studies have concluded that cigarette smokers are more likely to die at an earlier age compared to non-smokers and that this excess of deaths is largely due to CHD (Kannel, 1983; Kuller, Meilahn, & Ockene, 1985; Mann, Doll, Thorogood, Vessey, & Waters, 1976). The incidence of heart attack and sudden cardiac death is also directly related to the quantity of cigarettes smoked (Kannel, 1983; Kuller et al., 1985). Heavy smoking almost doubles the cardiovascular mortality of those under age 65 (Kannel, 1981). Despite the accumulation of information outlining the multiple hazards of cigarette smoking, approximately 41% of males and 32% of females in Canada still smoke (Health and Welfare Canada, 1981).

Age influences the detrimental effects of cigarette smoking. The greatest impact of this single risk factor is apparent in the age group with the lowest incidence of CHD (i.e., the youngest) with the effect decreasing as the disease incidence increases (Kannel, 1981). Thus, young smokers are more likely to suffer from a coronary event than older smokers when compared to their non-smoking counterparts.

Women who smoke appear to have less CHD than men who smoke (Kannel, 1981). Cigarette smoking has been shown to predispose women taking estrogen containing oral contraceptives to an increased risk of CHD (Mann et al., 1976). The risk of combining oral contraceptives with smoking is especially great in women over age 35 who have been using the pills for extended periods of time (Kannel, 1981).

HDL cholesterol, with its so-called "protective" effect has been found to be lower among smokers than non-smokers (Criqui, Wallace, Heiss, Mishkel, Schonfeld, & Jones, 1980; Garrison, Kannel, Feinleib, Castelli, McNamara, & Padgett, 1978; Goldbourt & Medalie, 1977; Williams, Robinson, & Baily, 1979). Significant differences of 0.08-0.1 mmol/l in men and 0.13-0.16 mmol/l in women have been measured (Garrison et al., 1978).

A person who stops smoking appears to substantially reduce the risk of CHD (Friedman, Petitti, Bawol, & Siegelau, 1981; Gordon, Kannel, McGee, & Dawber, 1974). In general, studies have not taken into account other predisposing factors, nor has it been established how soon the risk declines. Although some studies have suggested an immediate benefit of cessation (Gordon et al., 1974), others have indicated it may require 5-10 years for the CHD mortality of former smokers to decline to that of non-smokers (Doyle, Dawber, Kannel, Heslin, & Kahn, 1962; Hammond & Garfinkle, 1969). Rosenberg, Kaufman, Helmrich, and Shapiro (1985) suggested that the risk of MI in cigarette smokers decreased within a few years of quitting to a level similar to that of men who had never smoked. This result was not altered by allowance for other known predisposing factors for CHD.

2.4 Blood Pressure

Both systolic and diastolic blood pressures have been identified as important and independent risk factors for CHD (Dawber, 1980; Rabkin, Mathewson, & Tate, 1978; Rosenman, Sholtz, & Brand, 1976; Whelton & Russell, 1984). Pressures below 140/90 mm Hg have been widely recognized as normal, whereas, systolic pressures (SBP) of 140-159 mm Hg and diastolic pressures (DBP) of 90-94 mm Hg were considered to be in the borderline hypertensive range. Pressures consistently above 160/95 mm Hg were diagnostic of hypertension.

Using the categories of normotension, borderline, and hypertension for males and females participating in the Framingham study, regardless of age, the incidence of CHD increased as the pressure increased (Dawber, 1980). This relationship was true even when cases were grouped on the basis of a single casual measurement under usual office conditions.

The risk of developing CHD when associated with an elevated SBP was increased and the disease appeared at a younger age when compared to normotensives participating in the Framingham study (Dawber, 1980). DBP exhibited similar trends to SBP but the association was not as strong and the gradient of increase in disease incidence was not as steep (Dawber, 1980). This relationship was also found in other large scale investigations (Miall & Brennan, 1981; Rabkin et al., 1978; Rosenman, Brand et al., 1976).

Hypertension has been associated with a variety of other factors including age, family history, obesity, serum cholesterol and physical activity. The overall relationship between age and blood pressure has been rather consistent in western community-based surveys (Kannel & Gordon, 1978; Miall & Brennan, 1981; Rabkin et al., 1978; Roberts & Maurer, 1977; Rosenman, Brand et al., 1978). In most studies, blood pressure increased at a fairly constant rate in females, whereas in males the rate of rise during adolescence was faster but subsequently became less than females. The average SBP during adolescence and early adulthood was higher in males than females but this trend appeared to reverse in later life (Dawber, 1980). In both sexes, DBP rose more gradually with aging (Roberts & Maurer, 1977).

The tendency for an individual to stay roughly in the same rank of a distribution as he ages is known as "tracking" (Fixler, 1985). Numerous studies have shown that blood pressure tracking occurs in children (Berenson, 1980; Clarke, Schrott, Leaverton, Connor, & Lauer, 1978; Rosner, Hennekens, Kass, & Miall, 1977; Zinner, Margolius, Rosner, & Kass, 1978). Further studies have indicated that elevated blood pressure levels in adolescence were related to hypertension in adult-

hood (Harlan, Oberman, Mitchel, & Graybiel, 1973; Kuller, Crook, Almes, Detre, Reese, & Rutan, Paffenbarger, Thorne, & Wing, 1968; Sneiderman, Heyden, Heiss, Tyroler, & Hames, 1976). Webber Voors, Srinivasan, Frerichs, and Berenson (1979) suggested that the high degree of tracking of blood pressure in young people indicated that essential hypertension likely begins in childhood and potential hypertensive patients can be identified early in life.

Race has also influenced blood pressure levels. Adult blacks had higher mean SBP and DBP than their white counterparts as measured in a U.S. Health and Nutrition Education Survey (Roberts & Maurer, 1977). Children and adolescents have also exhibited a similar relationship (Berenson, 1980; Fixler, 1978).

Numerous investigations have confirmed the association between elevated blood pressure and obesity in adults (Epstein et al., 1965; Kannel, Brand, Skinner, Dawber, & McNamara, 1967; Kesteloot & VanHoute, 1974; Keys et al., 1972; Stamler, Stamler, Riedinger, Algera, & Roberts, 1978). Similar associations have been observed among adolescents and young adults in Evans County (Johnson, Cornoni, Cassel, Tyroler, Heyden, & Hames, 1975) and among youths in Bogalusa (Voors, Webber, Frerichs, & Berenson, 1977), Muscatine (Lauer, Connor, Leaverton, Reiter, & Clarke, 1975), and St. Louis (Goldring, Londe, Sivakoff, Hernandez, Britton, & Choi, 1977). The relationship between obesity and hypertension was also demonstrated in overweight subjects consuming a diet designed to lower body fat (Iacono, Dougherty, & Puska, 1982; Reisin, Abel, Modan, Silverberg, Eliahoi, & Modan, 1978; Tuck, Sowers, Dornfeld, Kledzik, & Maxwell, 1981).

2.5 Physical Activity

Physical activity is considered by some to be an important part of the multifactorial cardiovascular risk profile. It is thought to play a role by directly affecting the integrity of the cardiovascular system as well as by influencing the level of other major risk factors (Echner, 1983; LaPorte, Adams, Savage, Brenes, Dearwater, & Cook, 1984).

Several epidemiological studies have shown that the incidence of CHD is higher in persons who have inactive or sedentary occupations than in those who have active occupations (Cassel et al., 1971; Kahn, 1963; Morris, Heady, Raffle, Roberts, & Parks, 1953; Paffenbarger, Laughlin, Gima, & Black, 1970). Not all studies have found this association (Chapman & Massey, 1964; Paul et al., 1963; Pusnar & Karvonen, 1976). Certainly, job selection factors and the level of leisure time activity could have influenced these latter results.

Studies in which vigorous leisure time activity was accounted for were more likely to reveal a significant positive relationship. The epidemiological evidence suggests that endurance exercise protects against CHD (Paffenbarger & Hyde, 1984; Echner, 1983). Moderately intense activities such as walking, cycling, gardening, and stair climbing were found to be significantly associated with a lower risk of coronary disease (Magnus, Matroos, & Strackee, 1979; Morris, Everitt, Pollard, & Chave, 1980; Paffenbarger, Wing, & Hyde, 1978). Overall mortality, cardiovascular mortality, and CHD mortality in particular have been found to be inversely related to the level of physical activity in a variety of studies (Cooper, Meyer, Blide, Pollock, & Gibbons, 1977; Paffenbarger & Hyde, 1984; Wilhelmsen et al., 1981) but not all scientists have agreed (Bruce, DeRouen, Hossack, Blake, & Hofer, 1980). A low fitness level appeared to be a significant risk factor especially in those at high risk because of other cardiovascular risk factors (Kannel, Wilson, & Blair, 1985; Peters, Cady, Bischoff, Bernstein, & Pike, 1983).

Most studies have measured physical activity rather than physical fitness (i.e., maximal oxygen uptake). The question remains whether activity level or actual maximal oxygen uptake best predicts the risk for CHD (Froelicher, 1987). An examination of the epidemiologic studies of physical activity and CHD indicated that the high activity occupational groups generally did not achieve a sufficient intensity level to produce cardiovascular fitness (Goldsmith & Heile, 1971). Alternatively, there were groups that had high maximal oxygen consumption values but participated in only very limited activity (Astrand, Astrand, Hallback, & Kilbom, 1973). It is therefore suggested that activity levels and fitness are not necessarily synonymous in a population (LaPorte et al., 1984). Hickey, Mulcahy, Bourke, Bramhan, and Wilson-Davis (1975) suggested that the reduced CHD incidence associated with exercise might not be a direct effect of the exercise itself but a favourable modification of other well established risk factors.

Attempts have been made to quantify the amount of activity required to protect against the development of CHD (Morris, Chave, Adam, Sirey, Epstein, & Sheehan, 1973; Paffenbarger & Hyde, 1984; Paffenbarger et al., 1970; Rose, 1970; Shapiro, Weinblaff, Franck, & Sager, 1969; Skinner, Benson, McDonough, & Hames, 1966; Taylor et al., 1969). These estimates have been rather variable and ranged from about 500 kcal/week (Shapiro et al, 1969; Rose 1970) to approximately 2500 kcal/week (Skinner et al., 1966; Taylor et al., 1969). Paffenbarger and his group (1970) estimated that an increased expenditure of 924 kcal/day would yield a 34% reduction in CHD mortality.

The American College of Sports Medicine has made the following recommendations for the quality and quantity of exercise for developing and maintaining cardiorespiratory fitness and body composition. Four major components of activity are considered here: type, intensity, duration, and frequency.

Activities should be of the type that use large muscle groups and can be maintained for a prolonged period of time. They should be aerobic in nature. The intensity should correspond to 65-90% of maximal heart rate or 50-85% of maximal oxygen uptake. Conditioning periods should be 15-60 minutes of continuous or discontinuous aerobic activity. The duration is dependent upon the intensity of the activity, thus the lower intensity activity should be performed over a longer period of time. The frequency of conditioning should be 3 to 5 times per week (American College of Sports Medicine, 1978).

Several studies have shown a lower level of total cholesterol among those participating in vigorous physical activity when diet has remained relatively constant (Bjorntorp et al., 1972; Wood, Haskell, Klein, Lewis, Stern, & Farquhar, 1976). Physical training studies also show a reduction in this factor (Huttunen et al., 1979; Montoye, Block, Metzner, & Keller, 1976; Rounding, Fisher, & Anderson, 1978). On the other hand, numerous investigations have not shown any difference (Hurter, Peyman, Swale, & Barnett, 1972; Lehtonen & Viikari, 1978; Radliff, Elliott, & Rubenstein, 1978; Webster, Smith, LaRosa, Muesing, & Wilson, 1978; Wood et al., 1983). Some studies, however, did show a considerable reduction in mean cholesterol with exercise but statistical significance was not reached (Lopez, Vial, Balart, & Arroyave, 1974; Nye, Carlson, Kirsten, & Rossner, 1981). It is difficult to conclude what effect physical activity actually has on total plasma cholesterol levels.

Lipoprotein values in cross-sectional and longitudinal studies as they relate to physical activity have been fairly conclusive. Studies have consistently shown a higher mean HDL cholesterol level among physically active populations when compared to sedentary controls (Adner & Castelli, 1980; Carlson & Ericsson, 1975; Haskell, 1984; Martin, Haskell, & Wood, 1977; Wood & Haskell, 1979; Wood, Haskell, Stern, Lewis, & Perry, 1977). The investigations reporting lipoprotein changes with training covered a range of different groups, i.e., men and women, young

and old, and normal and hyperlipoproteinemic. A common pattern of change in plasma lipoprotein concentration is found in these reports. In general, HDL cholesterol increased while LDL cholesterol and VLDL cholesterol decreased (Huttunen et al., 1979; Lopez et al., 1974; Ratliff et al., 1978; Roundy et al., 1978; Webster et al., 1978; Wood et al., 1983).

It appears that relatively high levels of plasma TG can be considerably lowered by a program of vigorous physical activity (Holloszy, Skinner, Toro, & Cureton, 1964; Oscai, Patterson, Bogard, Beck, & Rothermel, 1972; Roundy, et al., 1978). Low initial levels of TG are affected in a less predictable manner (Huttunen et al., 1979; Lopez et al., 1974; Webster et al., 1978).

The level of physical activity has been shown to be associated with arterial blood pressure in cross-sectional surveys. In most studies, blood pressure has shown an inverse relationship to the level of occupational physical activity (Miall & Oldham, 1958; Montoye, Metzner, Keller, Johnson, & Epstein, 1972; Taylor, 1967). The level of physical fitness reveals a similar relationship (Cooper, Pollock, Martin, White, Linnerud, & Jackson, 1976; Gyntelberg & Meyer, 1974). Montoye and colleagues (1972) reported an even stronger inverse relationship when both occupation and leisure activities were considered. Not all studies, however, found this association (Doan, Peterson, Blackman, & Bruce, 1966; Pomrehn, Wallace, & Birmeister, 1982).

Longitudinal training studies also appear to provide evidence that physical training can help to lower blood pressure (Attina, Falorni, Canepile, Pieri, & Rossetti, 1981; Kasch & Kilberg, 1981; Penny, Rust, & Carlton, 1981; Pollock, 1973; Wilmore, Boyce, Girandola, Katch, & Katch, 1970). Subjects with the highest SBP values seem to gain the most benefit from an exercise program (Fagard, M'Buyamba, Staessen, Vanhees, & Amery, 1985; Wilmore et al., 1970).

2.6 *Body Composition*

The role of body composition as an independent risk factor for CHD has not been clearly established. Much of the early information used to implicate obesity as a risk factor came from actuarial data which suggested an increased total mortality among those who were overweight (Keys, 1981). These statistics have been criticized by the scientific community because, although including large numbers of insured lives, these groups consisted of selected subjects and therefore represented a biased sample (Bjorntorp, 1985).

The issue has been further confounded by using the term "overweight" synonymously with obesity. A person who is above the normal weight for a given height may be classified as overweight and therefore obese when he may simply have a greater muscle mass and not be "overfat" which is a more accurate term when defining obesity. Overweight should, therefore, be distinguished from "over fat" as being representative of obesity.

Numerous epidemiological investigations have used several different end points to indicate CHD including coronary death, angina pectoris, MI, or ECG findings which are considered to be reasonably diagnostic of MI or ischemia in order to search for a relationship between obesity and CHD. Several studies have found that measures of obesity have a predictive power for CHD only at the extreme upper end (i.e., most obese) of the population percentiles (Jarret, Shipley, & Rose, 1982; Noppa, Bengtsson, Wedel, & Wilhelmsen, 1980; Sorlie, Gordon, & Kannel, 1980). In contrast, other investigations have not supported the obesity/CHD hypothesis (Dyer, Stamler, Berkson, & Lindberg, 1975; Keys, 1980). Population differences, however, were apparent with the inclusion of people having health impairments and cultural differences. These investigations have had a duration of no longer than 18 years.

The Framingham population was re-evaluated after 26 years of study (Hubert, Feinleib, McNamara, & Castelli, 1983). The risk of CHD increased in both men and women having

increased Metropolitan Relative Weight (MRW) but the incidence was most pronounced in those younger than 50 years upon entry to the study. Similarly, among some 4000 Manitoba men also followed over a 26 year period, body mass index (BMI) was found to be related to the incidence of CHD (Rabkin, Mathewson, & Hsu, 1977). The association was most apparent in men less than 40 years old at entry to the study and was not evident until 16 years of follow up.

In persons that were followed for a sufficient length of time, it appeared that being obese at the time of entry into a prospective study was an independent risk factor predicting CHD and a reduced life expectancy. This does not include the possibility that obesity may generate, or be associated with other risk factors at a subsequent time (Simopoulos & Van Itallie, 1984).

Recent investigations have focused on the distribution of adipose tissue on the body. An excessive amount in the trunk region independent of total body fat has been shown to be associated with increased morbidity and mortality (Fitness and Amateur Sport, 1986). Statistically significant positive associations between the waist to hip circumference ratio and the occurrence of CHD were observed in a group of Swedish men (Larsson, Svardsudd, Welin, Wilhelmsen, Bjorn-
torp, & Tibblin, 1984). No differences were found between the risk of CHD and other indices of obesity such as the sum of skinfold thickness or BMI. The association with the waist to hip circumference ratio, however, was not significant in multivariate analysis when blood pressure and serum cholesterol concentration were taken into account.

Hubert and colleagues (1983) reported that in Framingham men, subscapular skinfold measurements were significantly and independently associated with an increased risk of MI whereas MRW was not. Similar results have been found elsewhere (Kissebah et al., 1982; Krotkiewski, Bjorn-
torp, Sjostrom, & Smith, 1983; Lapidus et al., 1984).

Obesity has been associated with the possibility of an increased prevalence of other cardiovascular risk factors. The incidence of hypertension in obese subjects was higher than that in the

non-obese, whether obesity was characterized according to relative body weight or skinfold thickness (Kannel et al., 1967; Noppa et al., 1980; Stamler et al., 1978). These relationships were also prevalent among young adults (Johnson et al., 1975).

Blair, Habicht, Sims, Sylwester, and Abraham (1984) examined the relationship between blood pressure and the distribution of subcutaneous body fat. Triceps and subscapular skinfolds were used as approximations of peripherally and centrally located body fat respectively. Subscapular skinfold appeared to be a better predictor of both SBP and DBP. The authors suggested that the blood pressure of middle-aged Americans was more directly associated with centrally deposited body fat although justification for the selected sites as being truly representative of central and peripheral body fat was not apparent.

Krotkiewski, Bjorntorp, Sjostrom, and Smith (1983) showed in a cross sectional study of men and women that cardiovascular risk factors such as hypertension and hypertriglyceridemia were more pronounced in subjects with a masculine type of adipose distribution (i.e., subjects with a high ratio of waist to hip circumference). Similar results were found by Kissebah and colleagues (1982).

Apparently, the relationship between obesity and CHD has been mediated by adverse changes in serum lipids and lipoproteins (Laskarzewski et al., 1980; Lauer et al., 1975; Webber et al., 1979) and blood pressure levels (Blair et al., 1984; Lauer et al., 1975; Webber et al., 1979) rather than obesity being an independent CHD risk factor variable.

2.7 *Family History*

Retrospective comparisons of the frequency of occurrence of CHD in first-degree relatives of CHD cases and controls has been made by a number of investigators (Deutscher, Ostrander, & Epstein, 1970; Forde & Thelle, 1977; Rissanen, 1979; Rose, 1964; Slack & Evans, 1966). Despite differences in nationality and methodology, certain results emerge consistently. Fathers of CHD patients have experienced more than twice the rate of CHD as have fathers of controls. The fathers of young CHD patients have an even greater relative risk for CHD. Mothers of patients have also shown a significant increase in CHD compared with mothers of controls in some but not all studies. Siblings may show even greater familial aggregation for CHD than do parents.

Prospective studies appear to confirm the retrospective findings (Gillum & Paffenbarger, 1978; Heller & Kelson, 1983; Sholtz, Rosenman, & Brand, 1975). These investigations also add other important details. Generally the rate of CHD tends to be highest and occurs at an earlier age in subjects whose parents developed CHD relatively early in life. This risk was increased when both parents developed CHD at a young age. The incidence of CHD was significantly related to the CHD experience of a sibling (Snowden, McNamara, Garrison, Feinleib, Kannel, & Epstein, 1982).

Numerous studies document the tendency for first-degree adult relatives of CHD patients to have more adverse risk factors than the general population (Deutscher et al., 1970; Feinleib, Kannel, Garrison, McNamara, & Castelli, 1976; Forde & Thelle, 1977; Glueck, Fallat, Tsang, & Buncher, 1974; Hamby, 1981; Morrison et al., 1980; Rissanen & Nikkila, 1977). This tendency was particularly striking in cases in which the affected relative manifested the disease at a young age. Increased rates of hypertension, hypercholesterolemia, hypertriglyceridemia, glucose intolerance, and obesity have been demonstrated repeatedly among the kin of such patients.

Some differences in risk factors are also evident among children with family histories of CHD although the relationship is unclear. Ibsen, Lous, and Andersen (1982) studied children and young adults whose fathers had died from ischemic heart disease before age 45. Hyperlipidemia was the only risk factor measured that was more common among case subjects. Stewart, Ratner, Goldberg, Johnson, & Gottlieb (1986) measured several risk factors in children age 12-19 whose parent had an MI or underwent coronary bypass surgery prior to age 50. Despite similar levels of aerobic fitness, obesity, and smoking habits, a less favourable lipid profile was found in female cases compared to controls. No differences were observed among the males.

Blonde, Webber, Foster, and Berenson (1981) and Shear, Webber, Freedman, Srinivasan, and Berenson (1985) investigated children (age 5-17) in the Bogalusa Heart Study whose parents had histories of MI, hypertension, diabetes, and/or stroke. Differences in some risk factors were evident and they appeared to be related to the specific parental disease (i.e., hypertensive children tended to have hypertensive parents).

It is clear that CHD and increased risk factors occur in families with a history of CHD. This aggregation inevitably reflects cojoined environmental and genetic effects (Rissanen, 1979; Rissanen & Nikkila, 1979). Some families, however, present evidence of genetic defects in the metabolism of cholesterol and its constituent subfractions. The most common of these is familial hypercholesterolemia. It is an inherited, autosomal dominant trait with a gene dosage effect, i.e., homozygotes are more severely affected than are heterozygotes. (Goldstein & Brown, 1983). The prevalence of heterozygotes among European, American, and Japanese populations is about 1 in 500 persons, whereas, homozygotes are much less common (1 in 1,000,000) (Goldstein, Schrott, Hazzard, Bierman, & Motulsky, 1973). The primary genetic defect in familial hypercholesterolemia results from one of several mutations in the gene specifying the receptor for plasma LDL resulting in a deficiency in LDL receptors (Goldstein & Brown, 1983). The receptor normally binds to LDL and facilitates cellular uptake and ultimate degradation of plasma LDL.

Familial hypercholesterolemia is characterized clinically by a selective elevation in the plasma level of LDL, deposition of LDL-derived cholesterol in tendons and arteries, and the inheritance of the LDL receptor defect (Goldstein & Brown, 1983). Heterozygotes have total plasma cholesterol levels in the range of 9.05-14.2 mmol/l made up mostly of LDL (Kwiterovich, Fredrickson, & Levy, 1974). The diagnosis of familial hypercholesterolemia does not simply depend on an elevation in plasma LDL but also includes demonstration of other factors relating to the disease (Goldstein & Brown, 1983). Cardiac disease commonly occurs in homozygotes between the ages of 5 and 30 (Khachadurian & Uthman, 1973). Few homozygotes survive past 30 years. Heterozygotes present evidence of CHD at an earlier age than the general population but not as early as homozygotes. For heterozygous men, the chances of having an MI by age 30 was 5%, and by the age of 50, 51% (Slack, 1969).

2.8 Occupation/Education

Several studies have generated reasonably consistent data indicating an inverse relationship between social status (as measured by occupation or educational attainment) and CHD risk. Rose and Marmot (1981), in a study of British civil servants, found that blue collar workers had an increased coronary risk over white collar workers. This was partly explained by the fact that blue collar workers smoked more, exercised less, were more overweight, and had higher blood pressures. Most of the difference, however, remained unexplained. Similar results, partially explained by life-style differences, have been found elsewhere (Hubert, Eaker, Garrison, & Castelli, 1987; Khoury et al., 1981; Kraus, Borhani, & Franti, 1980). It has been suggested that certain jobs that have been classified as blue collar are highly demanding but provide little decision making latitude and generally lead to job dissatisfaction (Karasek, Baker, Marxer, Ahlbom, &

Theorell, 1981; Karasek, Theorell, Schwartz, Pieper, & Alfredsson, 1982). Such job attributes have been associated with the development of CHD.

Several investigators have found that educational level is inversely associated with lifestyle related CHD risk factors (Donahue et al., 1985; Heiss et al., 1980; Liu et al, 1982; Rosenman, Brand, Sholtz & Friedman, 1976). The relationship remains similar when educational level is correlated with CHD incidence (Hinkle et al., 1968; Dawber, Kannel, Revotskie, Stokes, Kagan, & Gordon, 1959).

2.9 Summary

Conventional risk factors for CHD have been reasonably well delineated although the mechanisms of association remain unclear for most of the factors. Total cholesterol, HDL cholesterol, LDL cholesterol, smoking, blood pressure, and family history have all been well established in their relationship to the development of CHD. Physical activity, diet, triglycerides, alcohol consumption, and body composition are less well defined as predictors.

Middle-aged and older adults have been the most widely studied populations while several investigations in recent years have focused on children and adolescents. Comparatively little data currently exist on the levels of risk factors among the young adult population. It is poorly understood whether young adults exhibit similar relationships of lifestyle behaviours to CHD as do middle-aged and older adults.

Chapter III

METHODOLOGY

3.1 *Statement of the Problem*

The purpose of this investigation was to identify the elements which may affect the risk of CHD among young adults with a parental history of heart disease. Factors considered were the lifestyle habits of smoking, diet, and physical activity, and the physiological variables of total plasma cholesterol, HDL cholesterol, LDL cholesterol, triglycerides, blood pressure, and body composition. Young adult progeny of documented CHD patients were compared with similar subjects who did not present evidence of parental history of CHD.

3.2 *Hypotheses*

1. **Ho:** There is no difference in total plasma cholesterol between young adults with a CHD parent and young adults having parents without CHD.

Ha: Young adults with a CHD parent have higher total plasma cholesterol levels than young adults having parents without CHD.

2. **Ho:** There is no difference in HDL cholesterol levels between young adults with a CHD parent and young adults having parents without CHD.

Ha: Young adults with a CHD parent have lower HDL cholesterol levels than young adults having parents without CHD.

3. Ho: There is no difference in LDL cholesterol between young adults with a CHD parent and young adults having parents without CHD.

Ha: Young adults with a CHD parent have higher LDL cholesterol levels than young adults having parents without CHD.

4. Ho: There is no difference in triglyceride levels between young adults with a CHD parent and young adults having parents without CHD.

Ha: Young adults with a CHD parent have higher triglyceride levels than young adults having parents without CHD.

5. Ho: There is no difference in blood pressure between young adults with a CHD parent and young adults having parents without CHD.

Ha: Young adults with a CHD parent have higher blood pressure than young adults having parents without CHD.

6. Ho: There is no difference in the likelihood of smoking between young adults with a CHD parent and young adults having parents without CHD.

Ha: Young adults with a CHD parent are more likely to smoke than young adults having parents without CHD.

7. Ho: There is no difference in estimated body fat composition between young adults with a CHD parent and young adults having parents without CHD.

Ha: Young adults with a CHD parent have higher estimated body fat composition than young adults having parents without CHD.

8. **Ho:** There is no difference in dietary habits between young adults with a CHD parent and young adults having parents without CHD.

Ha: Young adults with a CHD parent are not as likely to follow recommended dietary habits with respect to CHD risk than young adults having parents without CHD.

9. **Ho:** There is no difference in physical activity levels between young adults with a CHD parent and young adults having parents without CHD.

Ha: Young adults with a CHD parent are less physically active than young adults having parents without CHD.

The null hypotheses will be rejected and the alternate hypotheses accepted if the variation among the sample proportions becomes larger than could be reasonably attributed to sampling error with $p < 0.10$.

3.3 Definitions

social interaction

Offspring have maintained verbal contact with parents by telephone or personal visits more than 6 times per year.

documented CHD

Persons having had one or more of the following significant events: myocardial infarction (MI), coronary artery bypass graft (CABG), or angioplasty.

3.4 Assumptions

1. Progeny of those having developed clinical CHD at a relatively early age are more likely to develop CHD themselves.
2. Social interactions between the young adult and his or her parents have been^d generally positive in nature.
3. A parent who was classified as not having documented CHD does not have underlying disease that has not yet been diagnosed.
4. Subjects fasted for a minimum of 14 hours prior to blood sampling as instructed.
5. A single blood pressure measurement was representative of each subject's usual value.
6. Data collected using a questionnaire were accurate to the best of the subject's knowledge.
7. Diet data collected were representative of the subject's usual habits and were accurately recorded.

3.5 Limitations

1. The accuracy of data collected by recall methods has low to moderate reliability, at best, and is subject to the limitations of the respondent's memory at the time.
2. A convenient sample was used in this study in order to obtain a sufficient number of subjects to carry out meaningful statistical analyses and, therefore, may not be truly representative of the young adult population.

3.6 *Delimitation*

1. Diet information in this study was limited to a food consumption report of three days in order to increase subject compliance. It has been estimated that nine days of food records are needed under free living conditions to estimate the correlation between dietary lipid intake and serum cholesterol with reasonable precision and avoid marked underestimation due to failure to control for intra-individual variability (Stamler, 1979a).

3.7 *Selection of Subjects*

All young adult subjects participating in this study were Caucasian and within the age range of 21-32 years. They were generally in good health and did not have any illness which may have altered either their lifestyle habits or physiological measures to any significant degree. Subjects who were pregnant at the time of investigation were excluded from the study.

Subjects participating in this study were from a sample of convenience. Control subjects were matched to case subjects on the basis of gender and occupation in order to reduce selection biases. All young adult participants provided their informed consent (Appendix A).

3.7.1 *Experimental subjects*

Case subjects were selected from the offspring of documented CHD patients. The first documented event of CHD was at least one year prior to the study. Subjects must have had some social interaction with their diseased parent. The CHD patients were contacted through local cardiac rehabilitation programs and their children were asked to participate in the study. Additional young adults with a parental history of CHD were identified when attempts were made to match cases to control subjects.

3.7.2 Control subjects

Control group subjects must have had at least one living parent with whom they had social interaction. Individuals were approached to participate according to gender and occupation once the case subjects had been selected. Both natural parents, whether living or dead, must not have had documented CHD. Subjects in this group with a step-parent having documented CHD were excluded from this investigation.

3.7.3 Both subject groups

Parents of the young adult subjects were asked to provide basic information on their medical history related to CHD. This information was confirmed by their physician wherever possible.

3.8 Independent and Dependent Variables

3.8.1 Independent Variable

parental history

Young adults were assigned to a test group based upon their parent's medical history. Case subjects had a parent with documented CHD. Parents of control subjects must not have had documented CHD.

3.8.2 Dependent Variables

total cholesterol (TC)

Total cholesterol was measured by the cholesterol oxidase method (Naito, 1984) and analyzed using the Perspective Analyzer (American Monitor Corporation).

triglycerides

Triglycerides were measured using the glycerol phosphate oxidase method (Foshati & Prencipe, 1982) and analyzed using the Perspective Analyzer (American Monitor Corporation).

HDL cholesterol (HDL)

HDL cholesterol was precipitated by the bioMerieux method (Naito, 1984). and loaded onto disks using an Autoloader (Electro-Nucleonics, Inc.) and analyzed in a Flexigem centrifugal analyzer (Electro- Nucleonics, Inc.).

LDL cholesterol (LDL)

LDL cholesterol was calculated using the subtractive method as follows (Naito, 1984): $LDL = TC - (TG \times 0.45 + HDL)$.

blood pressure Blood pressure was measured in the left arm following a 5 minute period of rest in a seated position. A Tycos Self Check Digital Blood Pressure Monitor (Model 7052-08) was used in order to reduce classification bias as much as possible. Values were coded as being normotensive, borderline, or hypertensive (see Table 2).

Table 2: Blood Pressure Score

SCORE	CATEGORY	SBP	DBP	COMMENTS
3	normal	<140	<90	both values within range
2	borderline	140-159	90-94	highest category of either SBP or DBP
1	high	>159	>94	

smoking

Questionnaire data (see Appendix B) were collected on current smoking habits and past smoking behaviours (Fitness and Amateur Sport, 1986).

Table 3: Body Composition Score

Percentiles and associated health risk zones by gender for body weight, adiposity, and fat distribution measures (age 20-29). (Fitness and Amateur Sport, 1986. Reproduced with permission.)

Measures		BMI		SOS		WHR		SOTS		
Gender		M	F	M	F	M	F	M	F	
SCORE	CATEGORY									Percentiles
4	at risk	19	18	26	37	76	65	13	13	95
1	above average	20	18	29	40	80	67	14	14	90
		21	19	30	43	81	68	16	16	85
		21	19	32	46	81	69	17	17	80
2	average	22	20	34	49	82	71	18	18	75
		22	20	36	51	83	72	19	19	70
		22	20	38	53	83	73	20	20	65
		23	21	40	56	84	73	21	21	60
		23	21	43	58	85	74	23	22	55
		23	21	46	60	85	75	25	23	50
3	below average	24	22	49	63	86	76	27	24	45
		24	22	52	65	87	76	28	26	40
		25	22	55	69	87	77	30	27	35
		25	23	58	72	88	78	32	29	30
5	at risk	26	23	62	76	89	78	35	31	25
		27	24	68	81	91	79	38	33	20
		27	25	74	86	93	80	41	36	15
		28	26	82	95	94	82	46	42	10
		30	28	94	111	96	85	54	48	5

Based on data from the Canada Fitness Survey, 1981.

**BMI Body Mass Index = Body Weight (kg) ÷ Height² (m)

SOS Sum of (five) Skinfolds (mm) = Triceps + Biceps + Subscapular + Iliac Crest + Medial Calf

WHR Waist to Hip Ratio = Waist Girth ÷ Hip Girth

SOTS Sum of (two) Trunk Skinfolds (mm) = Subscapular + Iliac Crest

Estimated Health Risk Zones According to Trends in Morbidity and Mortality Data

body composition

The method outlined in the Standardized Test of Fitness Operations Manual (Fitness and Amateur Sport, 1986) was used to assess body composition. This method takes into account a number of factors including body mass index (BMI), the sum of five skinfolds (SOS), the sum of trunk skinfolds (SOTS), and a waist to hip circumference ratio (WHR). Each of the four scores used to calculate body composition (BMI, SOS, WHR, SOTS) were divided into midrange values, above midrange values, and below midrange values after the "estimated health risk zones" (Fitness and Amateur Sport, 1986) were removed. Each value was then assessed a score from 1 to 5 with 1 being at least risk and 5 being at greatest risk (see Table 3) and summed into a body composition value. The possible range of values for body composition was 4 to 20. The raw scores found in Table 3 were used for all young adult subjects regardless of age.

physical activity

Details of leisure time physical activity involving the number of sessions, duration and intensity of activities participated in during the 4 weeks prior to study were collected using a questionnaire (Fitness and Amateur Sport, 1986). A self evaluation of the occupational work intensity was also included. Scores for physical activity were assessed based upon the American College of Sports Medicine and modified toward a more conservative end because of the difficulty in differentiating between physical fitness and physical activity as risk factors for CHD (see Table 4).

diet

A 3 day dietary report was used to assess levels of caloric intake, cholesterol, saturated and unsaturated fat. An instructional slide presentation was shown to each subject and a handout given prior to collection of diet data to facilitate reliability

Table 4: Physical Activity Score

SCORE	CATEGORY	CRITERIA
1	little	<12 activities of at least 15 minutes duration OR <8 activities of at least 15 minutes duration and at least of moderate intensity OR less than moderate occupational activity for most of the day with 4 leisure activities
2	moderate	12+ activities of at least 15 minutes duration OR 8+ activities of at least 15 minutes duration of at least moderate intensity OR at least moderate occupational activity for most of the day plus at least 4 leisure activities
3	heavy	16+ activities of at least 30 minutes duration at least 8 of which are at heavy intensity

of the measures. Nutrient Analysis (Behme, 1984), a microcomputer program package was used in this analysis. Foods reported that were not included in the Nutrient Analysis food list were obtained from Nutrients In Foods (Leveille, Rabik, and Morgan, 1983) and added to a foodfile list in the program. Diet was assessed on a point scale according to the recommendations of the American Heart Association and values that were typical of the North American diet (see Table 5).

Table 5: Conversion of raw diet data into diet scores

Values within the American Heart Association's recommended guidelines score 2 points. Values that are more typical of the North American diet score 1 point. Values that exceed these norms score 0 points. Points for each category are totalled to obtain a single diet score ranging from 0 - 8.

CATEGORY	POINTS AWARDED	
	2	1
% of total calories as fat	<30%	31%-40%
% of total calories as saturated fat	<10%	10.1%-16%
daily cholesterol intake	<300 mg	300.1-400 mg
body weight	3 of 4 scores contributing to body composition either 4 or 5 with no 1 or 2 scores except for BMI	N/A

3.9 Testing Protocol

Testing was conducted over a period of 2 months and subjects were evaluated on an individual basis or in small groups. Each subject was seated in a waiting area where the informed consent was provided (see Appendix A). A questionnaire related to medical history, physical activity, and smoking habits (see Appendix B) was completed at this time. The subject then viewed an instructional slide presentation outlining the information required for submitting a 3 day dietary report (Appendix C). Following this, a single blood pressure measurement was taken while the subject remained in a seated position.

The subject then proceeded to the testing area where several anthropometric measurements were taken including height, weight, waist (abdomen) and hip (gluteal) girth. Skinfolts were measured at the following standard sites: biceps, triceps, iliac crest, subscapular and medial calf.

Blood samples were collected from each subject following a 14 hour overnight fast. Samples were drawn from the brachial vein or another suitable surface vein in the arm. All procedures involving blood sampling and analyses were conducted under the supervision of certified laboratory staff at The Salvation Army Grace Hospital, Windsor. Before leaving the testing area, each subject was given an instruction package (see Appendix D) and a report form (see Appendix E) in order that food and drink consumption could be recorded for 2 week days and 1 weekend day. Report forms were returned to the investigator by mail in a self addressed envelope.

3.10 Statistical Analyses

The data were analyzed using the SPSSx computer system (SPSSx, 1988) with the alpha level set at 0.10 for all comparisons. The 90% confidence interval was used in order to reduce the potential of committing a Type II error by failing to reject the null hypothesis when it is false.

Several multiple analyses of variance (MANOVA) were used in order to examine differences in the relationships between several dependent variables at one time, thus reflecting real world situations. All dependent variables were pooled into a single analysis because of the multifactorial nature of the disease and the fact that victims of the CHD do not all present the same risk factors. Adverse lifestyle habits have been suggested as contributing factors to the development of CHD. Similarly, blood lipids, blood pressure, and smoking have been identified as critical factors and have, therefore, been included in separate analyses. One-way analyses of variance (ANOVA) were used to test the hypotheses in order to determine if there were differences

between each of the dependent variables on the independent variables of parental history of CHD and gender. The Pearson product moment correlation coefficient was used to measure the shared association of dependent variables and their component parts with one another.

Chapter IV

RESULTS

A sample of 56 volunteers was used in this study. Subjects were selected from young adults with or without a parental history of CHD and matched on gender and occupational group. Fourteen female pairs and 14 male pairs were included in the data analyses. None of these subjects had documented CHD or were being treated medically for CHD related disease. The physical characteristics of the young adult subjects are presented in Table 6.

A parent of the young adult participant was surveyed to determine appropriate group placement for the subject. Of the 46 parents contacted, 40 (87%) provided basic medical information which was confirmed by their physicians in 26 cases (65%). As a result, the accuracy of the assigned group remained unsubstantiated in 20 cases (36%). Six were non-respondents and 14 were unconfirmed by the physician (i.e., the physician was non-respondent). The assignment of a non-respondent's progeny to an experimental group was based upon the young adult's report of his parent's medical history. Parent data, including a gender breakdown of respondents and CHD history are found in Table 7. The mean age of onset of CHD among the parents was 51.8 years with a range of 44 to 66 years and a standard error of 1.35.

Multiple analyses of variance (MANOVA) failed to show differences in several different dependent variable combinations on the independent variable of parental history of CHD. The combinations of variables included all nine dependent variables as a group; the lifestyle variables of diet, physical activity, smoking, and body composition; blood lipids; and the major risk factors

Table 6: Physical characteristics of young adult subjects.

CHARACTERISTIC		MALES (n=28)	FEMALES (n=28)
age	mean	26.7 yrs	25.9 yrs
	range	21-32 yrs	21-32 yrs
	S.E.	.54	.53
height	mean	176.1 cm	167.3 cm
	range	163-195 cm	159-185 cm
	S.E.	1.3	1.2
weight	mean	79.5 kg	62.0 kg
	range	62-102 kg	47-107 kg
	S.E.	1.8	2.2
sum of five skinfolds	mean	70.2 mm	83.6 mm
	range	27.8-148.2 mm	49.8-194.0 mm
	S.E.	5.1	6.1
SBP	mean	121.4 mm Hg	107.1 mm Hg
	range	105-148 mm Hg	94-128 mm Hg
	S.E.	2.1	1.6
DBP	mean	77.0 mm Hg	71.1 mm Hg
	range	59-104 mm Hg	47-85 mm Hg
	S.E.	2.3	1.7

of smoking, blood cholesterol and blood pressure. Complete MANOVA results are presented in Table 8.

Analyses of variance (ANOVA) of the dependent variables (total cholesterol, HDL cholesterol, LDL cholesterol, triglycerides, blood pressure, smoking, body composition, physical activity, and diet) on the independent variable of parental history of CHD revealed a significant difference only in the triglyceride level. Young adults with a parental history of CHD had higher triglyceride levels than their counterparts without parental history of CHD ($p=.09$). All other

Table 7: Parent Characteristics

GROUP	GENDER	NUMBER SURVEYED	MI	CABG	ANGIOPLASTY
CHD History (n=18)**	males	16	12	4	0
	females	2	2	0	0
No CHD History (n=28)	males	14	0	0	0
	females	14	0	0	0

** Sibships were included among the offspring of this group.

Table 8: MANOVA results based on parental history

VARIABLE GROUPS	F-PROBABILITY
All dependent variables: smoking, BP, TC, HDL, LDL, TG, diet physical activity, body composition	.434
Lifestyle variables: diet, physical activity, smoking	.436
Lifestyle variables: diet, physical activity, smoking, body composition	.544
Blood lipids: TC, HDL, LDL, TG	.403
Major risk factors: BP, TC, LDL, smoking	.355

variables failed to reach the defined level of significance ($p < .1$). Complete ANOVA results are found in Table 9.

Table 9: ANOVA results based on family history

Analysis of variance table of nine dependent variables on the independent variable of family history.

DEPENDENT VARIABLES	PARENTAL CHD HISTORY	NO PARENTAL CHD HISTORY	F-RATIO	F-PROB.
BP score	$\bar{X}=2.8$ S.E.=.09	$\bar{X}=2.9$ S.E.=.05	1.80	.19
TC	$\bar{X}=5.24$ mmol/l S.E.=.18	$\bar{X}=5.01$ mmol/l S.E.=.16	.92	.34
LDL	$\bar{X}=3.41$ mmol/l S.E.=.16	$\bar{X}=3.28$ mmol/l S.E.=.16	.35	.56
HDL	$\bar{X}=1.27$ mmol/l S.E.=.05	$\bar{X}=1.24$ mmol/l S.E.=.07	.09	.76
TG	$\bar{X}=1.25$ mmol/l S.E.=.09	$\bar{X}=1.04$ mmol/l S.E.=.09	2.90	.09 *
Smoking score	$\bar{X}=1.3$ S.E.=.14	$\bar{X}=1.5$ S.E.=.22	.93	.34
Physical activity score	$\bar{X}=1.4$ S.E.=.11	$\bar{X}=1.6$ S.E.=.11	2.00	.16
diet score	$\bar{X}=3.1$ S.E.=.32	$\bar{X}=3.3$ S.E.=.37	.13	.72
body comp.	$\bar{X}=12.2$ S.E.=.66	$\bar{X}=12.7$ S.E.=.88	.21	.65

* $p < .1$

The results of this study allow rejection of only one null hypothesis. The alternative hypothesis #4 is accepted and indicates that young adults with a CHD parent have higher triglyceride levels than young adults having parents without CHD. The eight other null hypotheses could not be rejected at the $p < .10$ level of confidence.

Gender differences were apparent in the data (see Table 10). HDL cholesterol was higher among females than males ($p < .0001$) whereas LDL cholesterol was lower ($p = .04$). Total cholesterol and triglyceride differences across gender failed to reach significance although the trend of higher triglycerides among males was observed ($p = .12$). Females were more likely to consume a diet that was closer to the recommendations made by the American Heart Association ($p = .03$) and had higher (better) blood pressure scores ($p = .007$) when compared to the males. Females, however, were more likely to have lower estimated body composition scores (i.e., more CHD risk according to this factor) than males ($p = .01$). Significant differences were not shown in smoking behaviours or physical activity.

Interaction effects on the independent variables of parental history and gender were shown for smoking ($p = .075$) and systolic blood pressure ($p = .065$). Females with a parental history of CHD were less likely to smoke than those without such history, whereas males with a parental history of CHD were more likely to smoke when compared to their counterparts without parental history (see Figure 1). For systolic blood pressure, males had higher measures than females and those with parental history of CHD had SBP greater than those without such history for both genders. However, the difference in parental history among males was much greater than the difference among females (see Figure 2).

Correlation coefficients were computed for all dependent variables and their components. Although very few revealed an explained difference of greater than 49% ($r = .7$), many were nonetheless significant ($p < .1$). Appendix K lists all significant correlations that are meaningful (i.e., it does not include correlations for variable pairs that use similar raw data to make up the variable or component).

When comparing the group data as a whole to the recommended "ideal" values, it was found that 50% (28/56) of the subjects had total cholesterol values exceeding 5.17 mmol/l. LDL cholesterol was found to exceed 3.36 mmol/l in 45% (25/56) of subjects.

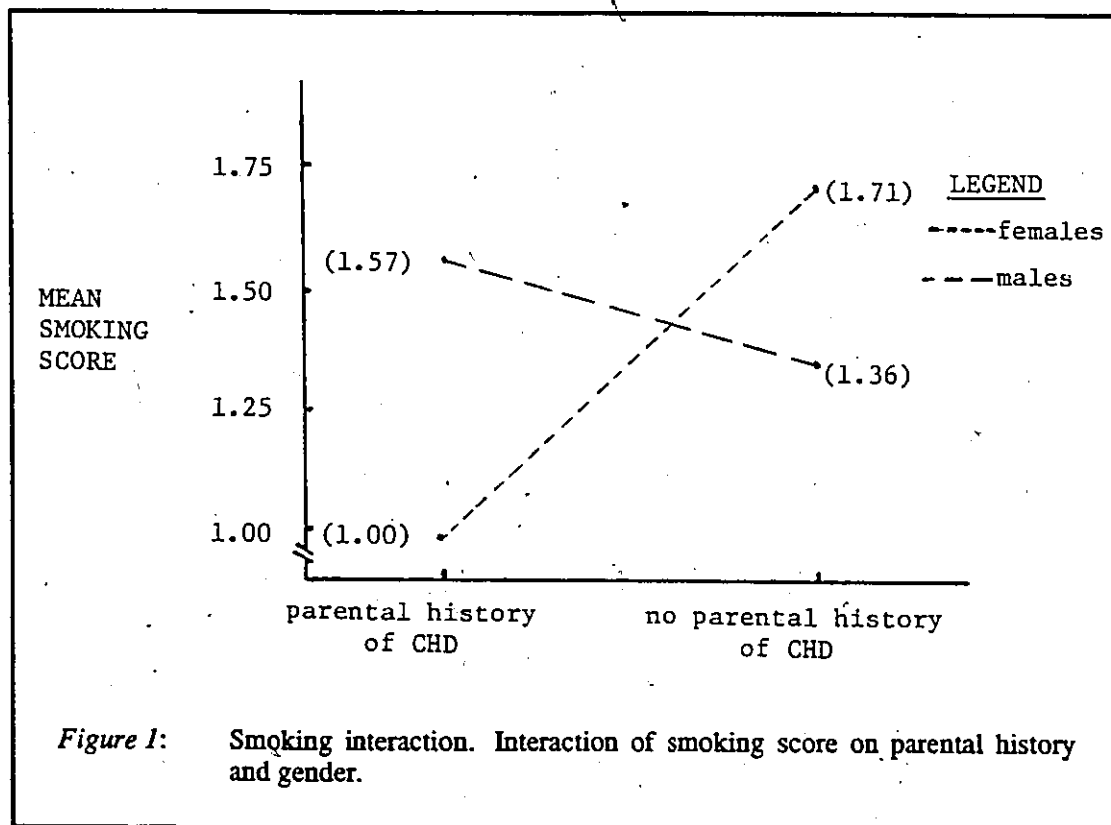
Table 10: ANOVA results based on gender

Analysis of variance table of nine dependent variables on the independent variable of gender.

DEPENDENT VARIABLES	MALES	FEMALES	F-RATIO	F-PROB.
TC	$\bar{X}=5.24$ mmol/l S.E.=.17	$\bar{X}=5.01$ mmol/l S.E.=.17	.96	.33
body comp. score	$\bar{X}=13.8$ S.E.=.77	$\bar{X}=11.1$ S.E.=.71	6.41	.01 *
HDL	$\bar{X}=1.09$ mmol/l S.E.=.04	$\bar{X}=1.42$ mmol/l S.E.=.06	21.81	<.0001*
LDL	$\bar{X}=3.57$ mmol/l S.E.=.16	$\bar{X}=3.12$ mmol/l S.E.=.15	4.31	.04 *
TG	$\bar{X}=1.25$ mmol/l S.E.=.10	$\bar{X}=1.04$ mmol/l S.E.=.08	2.54	.12
smoking score	$\bar{X}=1.5$ S.E.=.18	$\bar{X}=1.4$ S.E.=.19	.17	.68
diet score	$\bar{X}=2.6$ S.E.=.36	$\bar{X}=3.7$ S.E.=.30	4.93	.03 *
physical activity score	$\bar{X}=1.5$ S.E.=.12	$\bar{X}=1.5$ S.E.=.10	.22	.64
BP score	$\bar{X}=2.7$ S.E.=.10	$\bar{X}=3.0$ S.E.=.00	8.00	.007 *

* $p < .1$

Only one subject met all of the dietary recommendations of the American Heart Association for the purpose of modifying CHD risk factors (i.e., diet score = 8 points). Twenty percent of the subjects scored at least half of the maximum points (4 points), whereas 40% did not achieve more than 2 points (i.e., one quarter of the maximum 8 points).

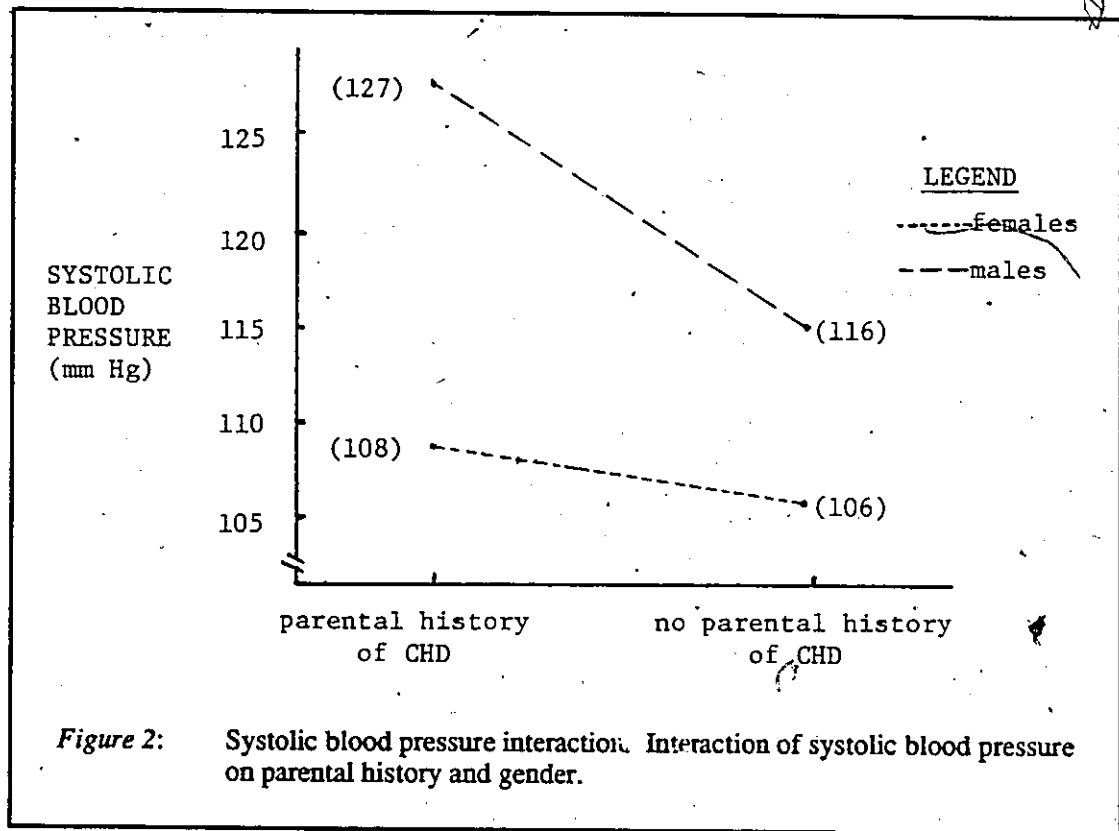


Of all 56 subjects, 46 (82%) were non-smokers. Within the group of 10 smokers, 2 were occasional smokers and 8 were regular smokers. Four of the smokers were female and 6 were male.

Blood pressure was within the normotensive range for 49 of the 56 subjects (87.5%). Six subjects (11%) were classified as borderline hypertensive and 1 (1.5%) was classified as hypertensive. Of those who did not fall within the normotensive category, all were male.

Physical activity scores revealed that 26 of 56 subjects (46%) were at least moderately active. Two of these subjects (both male) were classified in the "heavy physical activity" group.

Two of the subjects in the study scored maximum points (20) for body composition (i.e., above average but not at risk in all 4 measures). Fifty two percent of the subjects achieved at



least half of the points available. There were twice as many women as men who scored less than half of the available points.

Measures of reliability of blood lipids (total cholesterol, HDL cholesterol, and triglycerides) were found to be very high. Correlations on duplicated samples were at least .99.

Chapter V

DISCUSSION

The present study revealed increased levels of TG among young adults with parental history of CHD when compared to young adults without parental history of CHD. Other variables measured were not significantly different. This study partially agreed with the majority of the literature which documented the tendency for first-degree adult relatives of CHD patients to have more adverse risk factors than the general population (Deutscher et al., 1970; Feinleib et al., 1976; Forde & Thelle, 1977; Hamby, 1981; Morrison et al., 1980; Rissanen & Nikkila, 1977). These studies, however, used populations which were somewhat older than that used here (mean age of 45-50 years compared to a mean age of 26.2 years in this study) and who, therefore, may have had a greater separation of some risk factor levels among those with CHD (or underlying CHD) and those without CHD.

Several studies have found that children and adolescents with a parental history of CHD have increased levels of some risk factors (Blonde et al., 1981; Glueck et al., 1974; Ibsen et al., 1982; Shear et al., 1985; Stewart et al., 1986). The affected factors among children were reflected mainly in lipid and lipoprotein levels and in blood pressure although not all factor differences were found in all studies. It can thus be concluded that this study is somewhat supportive of the literature available to date regarding risk factors in a younger population relative to parental CHD.

It is difficult to determine the relative importance of the difference in TG levels among young adults with differing parental history found in this study. The majority of the literature failed to clearly report that elevated TG levels alone, placed a person at increased risk for the development of CHD. Strong recommendations for TG screening and treatment continue to appear despite this trend in the scientific literature (Bierman, 1980; Glueck, McGill, Shank, & Lauer, 1978; Tzagournis, 1978). The controversy appeared to arise from the correlation between TG and TC (Hulley et al., 1980, $r=.40$; this study, $r=.35$) and was based on the lipoprotein transport mechanisms they shared (Grundy, 1978). The analytic results of data from the Western Collaborative Group Study showed that cholesterol remained a strong predictor of CHD after adjustment for TG but that TG were no longer a significant predictor after adjustment for cholesterol (Hulley et al., 1980).

From a clinical standpoint, efforts to lower serum TG levels may also lower cholesterol levels (Grundy, 1978; Steinberg, 1979). Such interventions may be prescribed for the intrinsic merit of their influence on the highly atherogenic LDL cholesterol (Hulley et al., 1980). A fat and calorie controlled diet has appeared adequate as a general approach to hyperlipidemia (Connor & Connor, 1977; McGandy, Hegsted, & Stare, 1967; Wilson, Hulley, Burrows, & Nichaman, 1971). In a practical sense, then, it does not appear that knowledge of the TG level was needed for the selection of a treatment to lower serum cholesterol (Hulley et al., 1980).

The possibility remains that the TG level is important in development of CHD but the mechanisms of such risk are, as yet, unexplained. Certainly, it seems, that in white women over the age of 50, the TG level is an important factor (Heyden et al., 1980; Kannel et al., 1971; Lapidus et al., 1985).

It is interesting to note that grouping of the risk factors (dependent variables) in different combinations failed to reveal significant differences when examined by multivariate analyses. It

is possible that the disease process may not necessarily be reflected consistently among the specific variables or combinations of variables measured here.

One of the major differences between many of these studies and the present one is the differing definitions of parental disease which make direct comparisons difficult. Some studies have included hypertension, stroke, and diabetes mellitus and did not include categories of coronary artery bypass graft or angioplasty as was done here. Also, with the exception of Stewart and his colleagues (1986), all of the aforementioned investigations were conducted on a very large scale with several hundred subjects. In general population studies using children and adolescents (Blonde et al., 1981; Shear et al., 1985), the proportion of parents with disease, however it was defined, was considerably smaller than the reported total number of subjects (2-26.5% and 4-19% respectively).

With a smaller scale study such as this, the magnitude of difference between the two young adult categories needed to be much larger in order to identify more factors that may have been subtly different. Similarly, the categorization criteria of parental disease was limited to the presence or absence of documented CHD. The researcher was unable to investigate factors among the parents that may have indicated underlying heart disease (i.e., abnormal lipid or blood pressure levels, occlusion of coronary arteries in relatively asymptomatic patients, etc.) and subsequently, possible differences among the young adult progeny.

It was possible that the sample size used in this study was limiting to the detection of potential differences between the two young adult groups and that a much larger sample size would have increased the sensitivity of the results. Future research in this area may also be aided by incorporating more levels of the independent variable of parental history of CHD or by increasing the distance between the levels. This may be accomplished by separating the parents with documented CHD into groups that are of lower risk (i.e., no angina, non-smoker, blood pressure and

blood lipids under control) and higher risk (i.e., angina, smoker, poor control of blood lipids and blood pressure) and also those who have not yet developed CHD but have significant risk factors. The sensitivity of this design may also be increased in subsequent investigations by including more dependent variables such as apolipoproteins and HDL cholesterol subfractions, as technology allows, and by using other factors in component parts or composite scores or some combination of the two. The number of hours per week of light, moderate, or heavy intensity exercise, as well as, a combined score of physical activity might be used in the statistical analyses. Similarly, the use of bioelectric impedance may provide additional information in the assessment of body composition.

Subject selection biases may have been introduced in this study because of the author's limitations in recruiting appropriate subjects. Several young adults in the parental history group were selected through their parents who were active participants in cardiac rehabilitation programs. As a result, the profile of the physical activity levels among the parents with CHD are likely to be biased toward more physical activity when compared to the overall population of adults with CHD (see Table 11). Consequently, the behaviour of their young adult progeny may be altered somewhat, although it is unknown if the parental habits had any influence on the young adult.

Table 11: Physical activity of CHD parents

PHYSICALLY ACTIVE*	CARDIAC REHABILITATION PROGRAM PARTICIPANT	LITTLE PHYSICAL ACTIVITY
2	8	8

TOTAL PHYSICALLY ACTIVE = 10

* at least moderate activity according to classification outlined in Table G but not participating in an organized cardiac rehabilitation program.

In a similar way, lifestyle behaviour changes could have occurred among the group of young adults with parental history of CHD simply by exposure to a life threatening episode in a close family member. The nature of the changes made, if any, is unknown. Subjects in the control group were not exposed to the same family crises and potential group differences may have been obscured.

The gender differences in blood lipid found in this study were generally in agreement with those found in other investigations. Total serum cholesterol levels were not significantly different between men and women. Similar results have been shown by Heiss and colleagues (1980) but not by Donahue and colleagues (1985) where higher total cholesterol levels were found among young adult males. Women had higher levels of HDL cholesterol and lower values of LDL cholesterol. These results were consistent with other studies (Donahue et al., 1985; Heiss et al., 1980; Hubert et al., 1987). The current study, however, did not show gender differences in TG levels as has been observed elsewhere (Donahue et al., 1985; Heiss et al., 1980). The few contrasts found in this study compared to other investigations may have been the result of true population differences and also the fact that this study combined women who used exogenous sex hormones with those who did not, whereas, some studies separated the two groups. Overall, however, in the current study, it appeared that young adult men had poorer lipid profiles when compared to young adult women.

Blood pressure was higher among males than in females as reflected by a lower BP score. This has been observed in other studies (Dawber, 1980; Hubert et al., 1987; Whelton & Russell; 1984).

Among the lifestyle variables, Hubert and colleagues (1987) reported poorer body composition (as reflected by BMI) among men. This disagrees with the results found here where a poorer body composition value was found among women compared to men. The fact that BMI in this

study was only one part of the body composition score may contribute to the disagreement with the results of Hubert and colleagues (1987).

The current study revealed no significant main effects of smoking by gender or parental history of CHD. A significant interaction effect, however, was noted where males were more likely to smoke and females less likely to smoke when they had parental history of CHD compared to their gender counterparts without such history. These results contradict the data presented by Ibsen and colleagues (1982) where both males and females from Copenhagen, age 18-29, whose fathers died from ischemic heart disease at a young age were both less likely to smoke than randomly selected reference groups (60% to 19% in males and 59% to 45% in females). There is a much smaller percentage of Canadians who smoke (41% of males and 32% of females, Health and Welfare Canada, 1981), however, it is unknown as to whether parental smoking habits or disease had any effect on the smoking habits of the young adult offspring. Undoubtedly, there are trends among North Americans toward non-smoking and that this change in habit has been observed more in males than in females (Millar, 1985). It may be that females in this study with parental history of CHD were more influenced by non-smoking trends than their male counterparts.

A significant interaction effect of SBP on gender and parental history of CHD was shown in this study. Males with parental history were more likely to have higher SBP than males without such history. Females showed a similar relationship but the gradient of difference was much smaller. It was again, unclear as to why this may occur. SBP may be an important marker for CHD risk in males with parental history of CHD.

The evaluation of body composition is subject to a considerable degree of error. The assumptions underlying the equations used for the prediction of body fat from simple anthropometric measurements appear to be not universally applicable (Fitness and Amateur Sport, 1986).

The concept of "overweight" vs. "overfat" has further clouded the issue by the inclusion in numerous studies of measures of obesity based upon height and weight without accounting for lean body mass. The distribution of body fat has also entered the debate. The resultant confusion in the literature is abundant (Blair, et al., 1984; Fitness and Amateur Sport, 1986; Hubert et al., 1983). Consequently, it has been suggested by an Ad Hoc Advisory Committee, convened by Fitness Canada in 1984, that one should assess body composition by taking into account a number of factors including skinfolds, anthropometric measurements and ratios (Fitness and Amateur Sport, 1986). The body composition score used in this study was an attempt to address these concerns. Of interest here is the fact that several of the components used to make up the body composition score were significantly correlated with one another (range of $r=.43$ to $r=.87$) even though very different raw measures were used. This has also been shown in other studies where WHR was significantly ($p<.05$) and positively associated with BMI and summed skinfold measures using differing sites as determined by the authors (Lapidus et al., 1984; Larsson et al., 1984). It can be concluded that the body composition components and the total score are reasonable measures of obesity and can, therefore, be discussed as a whole.

The literature states that obesity is associated with hypertension (Kannel et al., 1967; Noppa et al., 1980; Stamler et al., 1978). More recently, a significant ($p<.05$) positive correlation was found to exist between WHR and SBP (Lapidus et al., 1984; Larsson et al., 1984) and WHR and DBP (Larsson et al., 1984). Similarly, in this study, significant correlations of WHR to SBP and DBP were found ($r=.51$ and $r=.41$ respectively). Other skinfold measures (sites defined by each author) were significantly correlated with SBP and DBP (Blair et al., 1984; Smoak et al., 1987). Again, in this study, significant correlations of SOS and SOTS with SBP and DBP were measured (range $r=.29$ to $r=.44$). The association between obesity (as determined by body composition measures) and blood pressure is therefore confirmed in this young adult population.

The association between blood lipid levels and body composition has revealed significant ($p < .05$) correlations among several different combinations of measures in the literature. Total cholesterol has been positively correlated with WHR (Lapidus et al., 1984) and BMI (Donahue et al., 1985; Laskarzewski et al., 1980). In the present study, total cholesterol had no significant association with any of the body composition measures used.

TG and LDL cholesterol have been shown to be positively associated with WHR (Lapidus et al., 1984), BMI (Donahue et al., 1985; Laskarzewski et al., 1980) and trunk skinfolds (Kissebah et al., 1982). The current investigation was in agreement with most of these studies as the TG level was positively related to most body composition measures ($r = .19$ to $r = .35$) with the exception of SOTS. SOTS is a measure of trunk skinfolds and directly contrasts with the results of Kissebah and colleagues (1982). The reason for this disparity is unclear.

The correlation of LDL cholesterol with body composition measures revealed conflicting results in this study. LDL cholesterol was positively related to WHR ($r = .21$) and agrees with Lapidus and colleagues, but its association with the sum of skinfolds was inverse ($r = -.20$). Again, it is not understood as to why this contrast exists.

HDL cholesterol was inversely correlated with total body composition ($r = -.20$) and WHR ($r = -.53$) in this study. This relationship has also been confirmed elsewhere (Laskarzewski et al., 1980).


The lifestyle variables measured in this study (smoking, diet, and physical activity) showed few significant correlations to lipid values. Smoking was inversely correlated with HDL cholesterol ($r = -.19$). This relationship has been consistently found in several other studies (Criqui et al., 1980; Garrison et al., 1978; Goldbourt & Medalie, 1977; Williams et al., 1979). The only other lifestyle variable in this study found to be significantly associated with any lipid value was the relationship between diet and triglyceride level ($r = .24$). Other studies have reported on favoura-

ble lipid changes with changes in diet (Brown et al., 1984; Hjermann et al., 1979) but have failed to report changes in triglyceride levels. The large intra-individual variations in diets among those in free living North American populations (Stamler, 1979) may have contributed to the failure to observe significant associations between diet and lipid values in the study.

Physical activity has been associated with lower levels of total cholesterol (Bjorntorp et al., 1972; Huttunen et al., 1979; Montoye et al., 1976), LDL cholesterol (Huttunen et al., 1979; Lopez et al., 1974; Ratliff et al., 1978; Webster et al., 1978), TG (Holloszy et al., 1964; Oscai et al., 1972; Roundy et al., 1978), and higher levels of HDL (Adner & Castelli, 1980; Haskell 1984; Martin et al., 1977; Wood et al., 1977). These associations were not observed in this study. The gross classification of physical activity levels and the self reported nature of data collection may have contributed to the lack of association found here.

The mean levels of TC and LDL found in this study appear to be somewhat higher than those found elsewhere (Donahue et al., 1985; Hubert et al., 1987) in similarly aged populations. HDL values for men were lower than those observed in other studies whereas the values for women were quite similar (Donahue et al., 1985; Hubert et al., 1987). Values for TG measured in this study were slightly lower for men than in the Beaver County Study (Donahue et al., 1985) and slightly higher for women. True population differences may account for these differences along with the high proportion of young adults with parental history of CHD included in this study.

Many subjects participating in this study had risk factor values that were outside of the suggested "ideal" levels among blood lipids, diet, body composition, and physical activity, but blood pressure, for most, fell into the normotensive range. The proportion of smokers in this investigation was well below that found in the general population including all age groups (Health and Welfare Canada, 1981).



Chapter VI

CONCLUSIONS AND RECOMMENDATIONS

This study was based on the premise that individuals with parental history of CHD are at greater risk for developing CHD than those without such history. Given that the atherosclerotic process begins early in life, this study was designed to examine differences in commonly used cardiovascular risk factors among young adults with differing parental histories of CHD.

Higher TG levels were observed among young adults with parental history of CHD compared to those without such history. Significant differences were not observed with respect to any other factors including total cholesterol, LDL cholesterol, HDL cholesterol, blood pressure, smoking, physical activity, diet, and body composition. Several other studies have shown similar trends in TG levels, but many have also shown differences in other blood lipids and in blood pressure although inconsistencies are apparent. Multivariate analyses also failed to show clear differences among the parental history groups indicating no definitive pattern of increased risk among the variables measured. In general, however, first-degree relatives of CHD patients appeared to have more unfavourable risk factors than the general population and this was shown in this study by higher TG levels.

The significance of having higher levels of serum triglyceride remains unclear. Clinical practitioners still target an elevated TG level as being an important indicator of CHD risk but the epidemiologic literature fails to clearly implicate such a level as an independent factor in CHD. Tracking of the TG levels over time in individuals may be critical in assessing CHD risk. This may be especially important among females.

The differences in risk factor levels between men and women were generally consistent with those found elsewhere. Men were more likely to have poorer lipid profiles and higher blood pressure than women.

Certainly, more prospective data from long term investigations on CHD risk factors among families are needed to determine the relative importance of these factors in young adult populations. The next few years will be important in this task as the current longitudinal studies in Framingham, Bogalusa, and Evans County, Georgia continue to follow younger populations over time. It will be important to investigate large samples with differing levels of the independent variable of parental or family history. More standardized measures may be included in these studies. Recent technological improvements have greatly increased the reliability of plasma lipid values but physical activity, body composition, and diet remain difficult to investigate.

Meanwhile, public education will continue to be an important focus in reducing the incidence of heart disease. Clearly, the trends are beginning to change as more people move into non-smoking groups and become more conscious of diet and physical activity. This increased health awareness may also encourage people to have other important clinical factors such as blood lipids and blood pressure measured on a more regular basis. Given the multiplicity of factors that contribute to CHD, public education will need to continue to address the issue from many points of view.

Appendix A

CONSENT TO TESTING PROCEDURE

I hereby consent to act as a subject in the research project outlined below, and realize that I may voluntarily withdraw from the project at any time.

The testing involves filling out a questionnaire on my personal medical history, physical activity, smoking, alcohol consumption, and dietary habits. My height, weight, skinfolds, girth, and blood pressure will also be measured.

A small amount of blood will be drawn from my arm in order to measure some components of my blood that may be related to heart disease. There may be some minor bruising resulting from this procedure.

I understand that the purpose of this study is to examine risk factors for heart disease. I have been informed that my identity will not be revealed in the publication or presentation of this research study, but that I will have access to any or all data which pertains to my personal involvement in the study.

I hereby affirm that I have read and understood the contents of this form, and do agree to participate as a subject in the above outlined research study.

Dated this _____ day of _____, 19__.

Witness _____

Signature _____

Appendix B

YOUNG ADULT QUESTIONNAIRE

I.D.# _____

Check most appropriate answer. Be sure to answer all questions.

Have you ever had a heart attack?

____ yes, ____ no

Have you ever had heart bypass surgery?

____ yes, ____ no

Have you ever had angioplasty (balloon catheter in the heart)?

____ yes, ____ no

If you are female, do you take birth control pills?

____ yes, ____ no

Do you take medication for your heart, blood pressure, or cholesterol level?

____ yes, ____ no

If yes, list medication _____

Do you have regular medical examinations?

____ yes, ____ no

If yes, how often? _____ yearly
____ every 2 years
____ other (specify) _____

Does your job require regular medical examinations?

____ yes, ____ no, ____ not applicable

How long has it been since your last thorough medical examination?

____ within the past year
____ 1 to 3 years
____ more than 3 years but less than 5 years
____ 5 to 10 years
____ more than 10 years

How much do you smoke? (check all that apply)

- ☐ non-smoker
- ☐ cigarettes occasionally
- ☐ less than 1/2 pack of cigarettes daily
- ☐ 1/2 to 1 pack of cigarettes daily
- ☐ more than 1 but less than 2 packs of cigarettes daily
- ☐ 2 or more packs of cigarettes daily
- ☐ pipe or cigar occasionally
- ☐ pipe or cigar daily

If you are a non-smoker, did you ever smoke regularly?

☐ yes, ☐ no

If yes, how long ago did you stop? _____ months _____ years

Why did you stop? ☐ peer/parental pressure
☐ medical/health reasons
☐ did not enjoy the habit
☐ other (specify) _____

How often do you usually drink alcohol?

- ☐ I don't drink alcohol (skip next question)
- ☐ less than once a month
- ☐ 1 to 3 times a month
- ☐ 1 to 2 times a week
- ☐ 3 to 4 times a week
- ☐ 5 to 7 times a week

About how many drinks do you usually have at a time? (one drink is equal to 1 bottle of beer, or 1 small glass of wine, or 1 shot of liquor)

- ☐ one
- ☐ two or three
- ☐ four or five
- ☐ six or seven
- ☐ eight or more

Classify the level of physical activity required by your job or your usual daily activities. (check one)

- ☐ little activity
- ☐ some moderate activity
- ☐ moderately active during most of the day
- ☐ some heavy activity
- ☐ heavy activity throughout the day

How often do you participate in vigorous physical activities? (check one)

- ☐ no regular participation
- ☐ 2 or 3 times per month
- ☐ 1 or 2 times per week
- ☐ 3 or 4 times per week
- ☐ more than 4 times per week

Appendix C

OUTLINE OF INSTRUCTIONAL SLIDE PRESENTATION FOR DIET REPORT

This slide presentation will be shown continuously in the subject waiting area. Each subject will be instructed to watch it through at least once. The following is a list of the major points to be addressed during the presentation:

Introduction and overview of the report.

- 3 day food and drink consumption
- importance of accuracy
- assurance of confidentiality

What to include on report form.

- all food and drink consumed including meals, snacks, alcoholic beverages
- smaller food items that are added to larger items (i.e., salad dressing, ketchup, cream and sugar, sauces, gravies, salt, butter, or margarine, etc.)
- all items in a food made up of several items (i.e., in a ham sandwich there may be butter, lettuce, mustard, or cheese as well as the ham and bread)

Important details about a particular item.

- fat content of milk (skim, 2%, homo)
- type of margarine or oil used (if labelled)
- method of food preparation (i.e., fried, baked, broiled, barbecued, boiled, microwaved, steamed, etc.)

Quantity of food items.

- visual representations of common measures for comparison (i.e., cup, teaspoon, tablespoon, etc.)
- convenient measurement terms

How to return report form.

Appendix D

3 DAY DIET RECORD INSTRUCTIONS

Please record all food and drink consumed during 3 days. You may choose any day you wish but try to select days that reflect your usual dietary habits. One day should be on the weekend and 2 days during the week. It is critical to the outcome of this investigation that your report be as accurate as possible. Do not leave anything out. Please note that you have been identified by a number to ensure that the information contained in the report remains confidential. Your report will not be compared or judged in and way except for the specific purposes of this investigation.

The accuracy of this report can be maintained by following the guidelines listed here:

1. Record all food and drink items including meals and snacks for each period of 24 hours.
2. The order of recording foods does not matter.
3. If foods are cooked, fried, or stir-fried in oils, include type of oil (corn, safflower, olive, etc.) if possible.
4. If margarine is used and it is labelled with the percentage of polyunsaturated fat or saturated fat, please include this in your report.
5. Include as much information about the food you consume as possible including the amount in common measures. For example:

FOOD ITEM	AMOUNT
milk, 2%	1 cup
ham sandwich: white bread	2 slices
ham	1 slice
butter	1 pat
prepared mustard	1/2 tsp.
ground beef patty, lean	1 med.
chocolate chip cookies	5 med.
banana	1 med.
grapefruit juice	1 cup
cream of mushroom soup, canned	2 cups
doughnut, chocolate covered	1 med.
Italian salad dressing, calorie reduced	2 tbsp.

6. Don't forget to include things like:

- mayonnaise, butter or margarine
- salad dressing
- sauces, gravies, etc.
- ketchup, relish, etc.
- whipped toppings
- sugar (or substitute) and milk or cream in tea, coffee, or on cereal
- pickles, olives, etc.
- peanut butter

7. If possible, include the method of preparation. For example:

FOOD ITEM	AMOUNT
chicken breast, fried	1/2
potatoes, boiled	2 medium

8. If salt is added to food while cooking or if you use additional salt at the table, please note this information.

9. Alcoholic beverages must also be included.

10. If you eat at a "fast food" outlet (Wendy's, McDonalds, etc.) include the name of the establishment in addition to what you ate or drank there.

11. For your convenience, use the following common measures and abbreviations wherever appropriate to indicate size of serving:

1 cup (c) = 8 fluid ounces (oz) = 250 ml
1 teaspoon (tsp)
1 tablespoon (tbsp)
1 pound (lb) = 1/2 kilogram (kg)
1 pat (for butter)
1 cube (for sugar)

Appendix E
DIET REPORT FORM

I.D.# _____ Day _____

FOOD ITEM	AMOUNT	CODE#	GRAMS
_____	_____	_____	_____
_____	_____	_____	_____
_____	_____	_____	_____
_____	_____	_____	_____
_____	_____	_____	_____
_____	_____	_____	_____
_____	_____	_____	_____
_____	_____	_____	_____
_____	_____	_____	_____
_____	_____	_____	_____
_____	_____	_____	_____
_____	_____	_____	_____
_____	_____	_____	_____
_____	_____	_____	_____
_____	_____	_____	_____
_____	_____	_____	_____

NOTE: PLEASE RETURN AS SOON AS POSSIBLE TO:
DEPARTMENT OF KINESIOLOGY
UNIVERSITY OF WINDSOR

Appendix F
PARENT INFORMATION

Name _____

Address _____

Postal Code _____

Home Phone _____

Work Phone _____

Date of Birth ____/____/____ (da/mo/yr)

Sex (circle) M F

Have you ever had a heart attack?

____ yes, ____ no

____ If yes, date _____

Have you ever had heart bypass surgery?

____ yes, ____ no

____ If yes, date _____

Have you ever had angioplasty (balloon catheter in the heart)?

____ yes, ____ no

____ If yes, date _____

In order that we may verify the above medical information, please provide the name and address of your physician and sign the release form at the bottom of the attached page.

Physician _____

Address _____

Have you recently (within the past year) participated in any of the following programs regarding heart health education or rehabilitation exercise? (check all that apply)

- ☐ University of Windsor Cardiac Exercise Program
- ☐ Y Heart Club
- ☐ Heart to Heart
- ☐ Cooking for a Healthy Heart
- ☐ other (specify) _____
- ☐ I have not participated in any programs recently.

How much do you smoke? (check all that apply)

- ☐ non-smoker
- ☐ cigarettes occasionally
- ☐ less than 1/2 pack of cigarettes daily
- ☐ 1/2 to 1 pack of cigarettes daily
- ☐ 1 to 2 packs of cigarettes daily
- ☐ more than 2 packs of cigarettes daily
- ☐ pipe or cigar occasionally
- ☐ pipe or cigar daily

How often do you participate in vigorous physical activities?

(check one)

- ☐ no regular participation
- ☐ 2 or 3 times per month
- ☐ 1 or 2 times per week
- ☐ 3 or 4 times per week
- ☐ more than 4 times per week

Indicate the physical activities in which you have participated over the last month during your leisure time.

Fill out the following chart using these guidelines:

Frequency - number of occasions over the last month

Duration - average number of activity minutes spent on each occasion

Intensity - **LIGHT** - slight change from normal state

MEDIUM - some perspiration, faster than normal breathing

HEAVY - heavy perspiration, heavy breathing

ACTIVITY	FREQUENCY	DURATION				INTENSITY		
		1-15	15-30	31-60	60+	Light	Med	Heavy
walking	_____	_____	_____	_____	_____	_____	_____	_____
swimming	_____	_____	_____	_____	_____	_____	_____	_____
cycling	_____	_____	_____	_____	_____	_____	_____	_____
jog/run	_____	_____	_____	_____	_____	_____	_____	_____
tennis	_____	_____	_____	_____	_____	_____	_____	_____
squash	_____	_____	_____	_____	_____	_____	_____	_____
dancing	_____	_____	_____	_____	_____	_____	_____	_____
exercise class	_____	_____	_____	_____	_____	_____	_____	_____
others (specify)	_____	_____	_____	_____	_____	_____	_____	_____
_____	_____	_____	_____	_____	_____	_____	_____	_____
_____	_____	_____	_____	_____	_____	_____	_____	_____
_____	_____	_____	_____	_____	_____	_____	_____	_____

Classify the level of physical activity required by your job or your usual daily activities.

- _____ little activity
 _____ some moderate activity
 _____ moderately active during most of the day
 _____ some heavy activity
 _____ heavy activity throughout the day

Your adult children living in the Essex County region with whom you have some social interaction will be the primary data sources in this investigation. Please provide us with the following information so that we may contact them. Do not include the names of estranged children.

Name _____ Age _____ years

Address _____

Postal Code _____

Phone _____

Name _____ Age _____ years

Address _____

Postal Code _____

Phone _____

Name _____ Age _____ years

Address _____

Postal Code _____

Phone _____

Appendix G

PHYSICIAN LETTER

Dear Dr.

Re: Patient's name _____
D.O.B. _____
Code # _____

The above patient wishes to participate in a heart disease study conducted in the Department of Kinesiology as part of a graduate thesis. Briefly, we wish to look at risk factors among young adults with different family histories of heart disease. Your verification of medical information is necessary to ensure the validity of this investigation. You will note the bottom of this page has been signed by the patient and releases this information to us. The patient has been given a code number and file data will be kept strictly confidential.

Please provide us with the information outlined on the attached form at your earliest convenience.

Thank you for your assistance.

Sincerely,

Linda Stanczak,
Graduate Student.

Dr. Ray Hermiston,
Faculty Advisor.

Please provide the University of Windsor CHD study with information regarding significant occurrences related to my heart.

Patient's signature _____

Appendix H

PHYSICIAN VERIFICATION OF MEDICAL HISTORY

Patient Code # _____

This patient has had the following:

_____ myocardial infarction	date _____
_____ coronary artery bypass surgery	date _____
_____ angioplasty	date _____
_____ exercise induced angina	
_____ unstable angina	
_____ angiography indicating significant narrowing of the coronary arteries	
_____ none of the above	

Physician's signature _____

Dated _____

Thank you for your assistance.

Please return this page only to:

MRS. LINDA STANCZAK
DEPARTMENT ON KINESIOLOGY
UNIVERSITY OF WINDSOR
WINDSOR, ONT.
N9B 3P4

Appendix I

YOUNG ADULT SCREENING QUESTIONNAIRE

GENERAL DATA

Name _____

Address _____

Postal Code _____

Home Phone _____

Date of Birth ____/____/____ (day/mo/yr)

Sex (circle) M F

If female, are you pregnant? ____ yes, ____ no, ____ don't know

Race (check one only)

____ white (Caucasian)

____ black (Negro)

____ mulatto (1 Caucasian parent, 1 Negro parent)

____ Asian

____ other (specify) _____

Occupational Group (check one only)

____ professional (specify) _____

____ manager/supervisor

____ skilled trades

____ labourer

____ clerical

____ service (i.e., police, waiter, hairdresser, store clerk, etc.
(specify) _____

____ student - program _____

____ homemaker

____ unemployed

____ other (specify) _____

Education (check highest level)

Did you graduate?

	yes	no	currently enrolled
<input checked="" type="checkbox"/> elementary school	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
<input type="checkbox"/> secondary school	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
<input type="checkbox"/> trade school	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
<input type="checkbox"/> community college	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
<input type="checkbox"/> university	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>

Do you reside with your parent(s)?

☐ yes, ☐ no

What is your marital status?

☐ single
☐ married
☐ common-law
☐ separated
☐ divorced
☐ widowed

Do you have any children (natural or adopted)?

☐ yes, ☐ no

If yes, give ages _____

Indicate the frequency of social contacts (i.e., phone conversations, personal visits) you have with your parent.
 (check one)

☐ more than once a day
☐ 4 to 7 days per week
☐ 1 to 3 days per week
☐ 3 to 4 times per month
☐ 1 to 2 times per month
☐ 6 to 12 times per year
☐ less than 6 times per year

PERSONAL MEDICAL HISTORY

Have you ever had any serious health disorder? (check all that apply)

- ☐ cancer
☐ diabetes
☐ liver problems
☐ bone or joint disease
☐ rheumatic fever
☐ stomach or intestinal disorder
☐ other (specify) _____
☐ no serious health problems

FAMILY HISTORY

The following 3 questions deal with the health of your parent(s). Indicate his/her age at the time of heart attack or surgery.

Have either of your natural parents ever had a heart attack?

- ☐ no ☐ yes (father). Age at first heart attack _____ years
☐ ☐ yes (mother). Age of first heart attack _____ years
☐ don't know

Have either of your natural parents ever had heart bypass surgery?

- ☐ no ☐ yes (father). His age at time of surgery _____ years
☐ ☐ yes (mother). Her age at time of surgery _____ years
☐ don't know

Have either of your natural parents ever had angioplasty (balloon catheter in the heart)?

- ☐ no ☐ yes (father). His age at time of surgery _____ years
☐ ☐ yes (mother). Her age at time of surgery _____ years
☐ don't know

Does your mother or father participate in a cardiac rehabilitation or education program?

- ☐ yes (specify program) _____
☐ no
☐ don't know

Do you have a step-parent?

☐ no (skip to last item)

☐ yes - Please answer the following questions.

Have either of your step-parents ever had:

☐ heart attack

☐ heart bypass surgery

☐ angioplasty (balloon catheter in the heart)

☐ don't know

Do either of your step-parents participate in a cardiac exercise or education program?

☐ yes (specify program) _____

☐ no

☐ don't know

It may be necessary to contact your parents to confirm the above data. Please provide the following information:

Father's name _____

Address _____

Postal Code _____

Phone _____

Mother's name _____

Address _____

or same as father's _____

Postal Code _____

Phone _____

Appendix J

RAW DATA

PARENTAL HISTORY OF CHD - MALE

TC	HDL	TG	LDL	SBP	DBP	SMOKING SCORE	PHYSICAL ACTIVITY
5.12	1.02	2.24	3.09	140	094	1	1
6.43	1.42	1.09	4.52	128	092	1	1
3.85	1.17	0.68	2.37	126	081	1	1
4.71	1.07	0.74	3.31	123	068	1	1
6.18	1.14	2.02	4.13	115	063	1	2
6.24	0.93	2.50	4.20	116	061	1	1
4.49	1.26	0.93	2.81	148	091	3	1
5.49	1.26	2.00	3.33	134	104	4	2
4.96	1.30	0.86	3.27	132	066	1	1
4.14	0.90	1.13	2.73	105	073	2	1
5.32	0.94	1.23	3.83	119	082	3	2
7.14	1.03	0.97	5.70	118	059	1	3
4.84	0.94	1.06	3.42	132	085	1	1
4.38	0.96	1.41	2.79	144	073	1	2

BMI	SOS	WHR	SOTS	FAT	SFAT	CHOL	BW
31.6	148.2	0.89	91.2	43	13.8	138.0	0
23.8	058.4	0.81	35.8	42	13.9	347.0	0
22.8	053.8	0.77	26.0	46	15.9	358.3	0
22.0	041.8	0.77	22.6	37	12.3	218.3	1
24.2	069.2	0.87	33.0	51	14.3	310.0	0
23.6	124.4	0.85	67.4	43	15.0	590.0	0
24.9	064.4	0.81	36.2	45	14.4	446.5	0
30.3	098.6	0.88	66.2	37	12.2	251.3	0
24.8	048.4	0.82	24.4	34	10.2	416.0	1
24.6	065.6	0.84	35.8	42	16.8	380.0	0
22.6	043.8	0.88	24.2	55	16.2	999.9	1
26.6	035.8	0.84	23.4	50	20.3	538.0	1
25.1	088.8	0.85	52.4	31	09.4	212.0	0
25.8	067.1	0.82	38.4	36	13.2	546.0	0

FAT = % dietary fat

SFAT = % dietary saturated fat

CHOL = amount of dietary cholesterol (mg)

BW = body weight score

NO PARENTAL HISTORY OF CHD - MALE

TC	HDL	TG	LDL	SBP	DBP	SMOKING SCORE	PHYSICAL ACTIVITY
5.53	1.12	0.96	3.98	119	094	1	1
5.65	1.19	1.04	3.99	118	059	1	2
6.87	0.73	1.52	5.46	117	074	4	2
5.40	1.04	1.03	3.90	114	074	1	1
4.30	0.99	1.32	2.72	107	070	1	3
5.06	0.97	2.27	3.07	109	074	1	1
5.20	1.13	1.07	3.60	112	078	1	2
4.99	1.55	0.56	3.19	127	083	1	1
5.28	1.16	0.52	3.89	124	068	1	2
3.84	1.35	1.30	1.91	114	062	3	2
6.05	1.01	1.72	4.30	121	078	1	2
6.44	1.22	1.03	4.76	107	071	1	1
4.57	0.81	0.86	3.37	116	091	1	1
4.34	0.79	0.84	3.17	115	087	1	2

BMI	SOS	WHR	SOTS	FAT	SFAT	CHOL	BW
25.8	062.4	0.87	33.2	42	14.4	860.0	0
23.6	027.8	0.78	13.8	42	14.7	251.3	0
29.4	054.8	0.87	37.8	44	17.0	249.3	0
25.3	079.2	0.91	54.8	34	12.6	196.3	0
24.3	095.0	0.83	55.8	33	09.6	148.0	0
21.7	045.4	0.82	21.6	42	15.2	634.3	1
27.0	056.6	0.84	28.8	58	15.1	220.7	0
26.0	076.4	0.88	38.0	42	17.9	510.7	0
22.9	049.0	0.75	23.4	23	09.8	220.0	1
27.8	088.4	0.93	55.6	47	18.3	580.0	0
25.1	075.2	0.83	42.8	48	14.8	324.7	0
25.3	072.2	0.80	39.2	52	18.4	495.0	0
34.1	108.2	0.98	72.0	48	12.7	560.7	0
26.8	066.6	0.89	38.4	32	10.2	106.3	0

FAT = % dietary fat

SFAT = % dietary saturated fat

CHOL = amount of dietary cholesterol (mg)

BW = body weight score

PARENTAL HISTORY OF CHD - FEMALE

TC	HDL	TG	LDL	SBP	DBP	SMOKING SCORE	PHYSICAL ACTIVITY
5.32	1.43	2.01	2.99	100	080	1	1
4.03	1.22	0.71	2.49	109	084	1	1
5.05	1.74	0.82	2.94	103	065	1	1
5.43	1.29	0.76	3.80	102	058	1	2
5.58	1.16	1.27	3.85	121	066	1	1
6.56	1.37	1.34	4.59	116	068	1	2
4.07	1.37	1.07	2.22	099	068	1	1
6.75	2.02	1.16	4.21	105	073	1	2
3.93	1.61	1.12	1.82	112	055	1	2
5.12	1.43	0.86	3.30	128	078	1	1
5.79	1.16	1.21	4.09	112	076	1	1
6.38	1.43	1.86	4.11	102	074	1	1
5.25	1.34	1.03	3.45	105	069	1	2
4.19	1.59	1.01	2.15	102	085	1	1

BMI	SOS	WHR	SOTS	FAT	SFAT	CHOL	BW
40.3	194.0	0.70	87.8	46	16.6	327.0	0
23.4	120.8	0.69	38.4	47	14.8	321.7	0
21.0	067.6	0.70	21.2	46	18.8	204.0	1
19.1	060.4	0.68	23.2	34	11.8	148.0	1
22.8	108.4	0.70	43.2	54	12.9	157.0	0
19.7	072.4	0.72	17.8	50	15.9	375.0	1
17.6	062.3	0.69	21.7	40	13.2	661.3	0
19.0	070.8	0.64	19.6	46	17.7	315.3	0
21.1	075.6	0.65	25.2	31	10.2	261.0	0
25.6	136.9	0.75	68.2	33	13.6	337.0	0
27.0	113.4	0.75	53.2	30	12.2	201.0	0
21.5	080.8	0.72	33.6	48	14.6	211.0	0
22.2	057.0	0.69	18.4	33	06.6	136.0	1
20.9	075.0	0.73	30.4	37	11.1	240.0	0

FAT = % dietary fat

SFAT = % dietary saturated fat

CHOL = amount of dietary cholesterol (mg)

BW = body weight score

NO PARENTAL HISTORY OF CHD - FEMALE

TC	HDL	TG	LDL	SBP	DBP	SMOKING SCORE	PHYSICAL ACTIVITY
3.78	1.07	0.94	2.29	108	071	4	2
3.31	1.24	0.85	1.69	110	078	1	1
5.60	1.62	1.38	3.36	126	073	2	2
4.96	1.35	1.00	3.16	104	066	1	2
5.34	2.49	0.52	2.62	098	081	1	2
4.54	1.10	0.92	3.03	094	047	3	1
3.97	0.78	0.84	2.81	112	076	1	2
5.35	1.71	0.45	3.44	103	061	1	1
5.58	1.47	1.92	3.23	109	075	1	1
6.17	1.72	1.24	3.89	114	074	1	1
5.40	1.17	0.54	3.99	098	079	5	1
4.57	1.37	0.44	3.00	095	065	1	1
4.31	1.55	1.37	2.14	110	077	1	2
3.91	1.07	0.57	2.58	103	069	1	2

BMI	SOS	WHR	SOTS	FAT	SFAT	CHOL	BW
25.4	088.4	0.70	47.6	51	23.3	345.0	0
19.2	049.8	0.70	19.6	47	16.7	194.0	1
24.4	118.6	0.71	56.0	39	10.6	234.0	0
19.1	052.6	0.70	18.8	42	11.8	209.0	1
39.1	103.8	0.73	47.6	34	13.7	227.0	0
18.0	055.0	0.67	16.8	45	15.9	348.0	1
23.8	077.0	0.70	31.6	47	13.5	209.0	0
20.0	060.8	0.70	19.0	54	19.3	218.7	1
24.7	063.8	0.74	19.6	42	13.9	220.0	1
20.4	052.4	0.68	17.4	44	16.9	333.0	1
21.3	055.2	0.70	17.0	39	14.5	233.0	1
20.2	067.0	0.67	22.6	36	12.1	150.0	1
21.8	104.2	0.70	47.4	33	11.0	362.0	0
23.1	095.8	0.74	45.4	41	14.3	347.7	0

FAT = % dietary fat

SFAT = % dietary saturated fat

CHOL = amount of dietary cholesterol (mg)

BW = body weight score

Appendix K

SIGNIFICANT CORRELATION COEFFICIENTS ($P < .1$)

	r Value
LIPIDS VS LIPIDS	
HDL - LDL	-.18
HDL - TG	-.19
LDL - TG19
LIPIDS VS BODY COMPOSITION VALUES	
HDL - total body composition (BCOMP)	-.20
HDL - WHR	-.53
LDL - sum of skinfolds (SOS)	-.20
LDL - waist to hip ratio (WHR)21
TG - BCOMP19
TG - BMI23
TG - SOS35
TG - WHR27
LIPIDS VS LIFESTYLE FACTORS	
HDL - smoking	-.19
TG - diet24
LIPIDS VS BLOOD PRESSURE	
HDL - SBP	-.27
TG - SBP19
TG - blood pressure score (BPS)	-.22
BLOOD PRESSURE VS LIFESTYLE FACTORS	
BPS - smoking22
BPS - diet18
DBP - physical activity score (PAS)	-.26
SBP - diet	-.18
DBP - diet	-.20

BODY COMPOSITION VS LIFESTYLE FACTORS

BCOMP	- diet	-.44
BMI	- diet	-.28
SOS	- diet	-.23
SOTS	- diet	-.32
WHR	- diet	-.37

BLOOD PRESSURE VS BODY COMPOSITION

BPS	- BCOMP	-.36
BPS	- BMI33
BPS	- sum of trunk skinfolds (SOTS)	-.36
BPS	- WHR	-.41
SBP	- BCOMP27
SBP	- SOTS30
SBP	- WHR51
DBP	- BCOMP45
DBP	- SOS29
DBP	- SOTS44
DBP	- WHR41
BMI	- SBP23
BMI	- DBP46

BODY COMPOSITION MEASURES

BMI	- SOS57
BMI	- SOTS71
BMI	- WHR49
SOS	- SOTS87
SOTS	- WHR43

BLOOD PRESSURE MEASURE

SBP	- DBP43
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